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ABSTRACT

This curriculum supplement guide brings the latest medical discoveries to classrooms. This module focuses on the objectives of introducing students to major concepts related to the development of cancer and its impacts, and developing an understanding of the relationship between biomedical research and personal and public health. This module includes five major sections: (1) "Understanding Cancer"; (2) "Implementing Module"; (3) "Student Activities"; (4) "Additional Resources for Teachers"; and (5) a glossary and references section. (Contains 26 references.) (YDS)

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Cell Biology and Cancer

National Institutes of Health

National Cancer Institute

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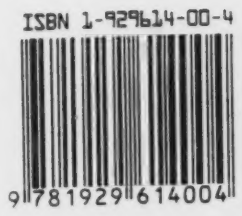

Cell Biology and Cancer

under a contract from the
National Institutes of Health

National Cancer Institute

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Foreword

This curriculum supplement brings into the classroom new information about some of the exciting medical discoveries being made at the National Institutes of Health (NIH) and their effects on public health. This set is being distributed to teachers around the country free of charge by the NIH to improve science literacy and to foster student interest in science. These tools may be copied for classroom use, but may not be sold.

This set was developed at the request of NIH Director Harold Varmus, M.D., as part of a major new initiative to create a curriculum supplement series (for grades kindergarten through 12) that complies with the *National Science Education Standards*.[1] This set is part of a continuing series being developed by the NIH Office of Science Education (OSE) in cooperation with NIH institutes with wide-ranging medical and scientific expertise. Three new supplements are planned per year.

The curriculum supplements use up-to-date, accurate scientific data and case studies (not contrived). The supplements contain extensive background information for teachers and

- use creative, inquiry-based activities to promote active learning and stimulate student interest in medical topics;
- deepen students' understanding of the importance of basic research to advances in medicine and health;
- offer students an opportunity to apply creative and critical thinking;
- foster student analysis of the direct and indirect effects of scientific discoveries on their individual lives and on public health; and
- encourage students to take more responsibility for their own health.

Each supplement contains several activities that may be used in sequence or as individual activities designed to fit into 45 minutes of classroom time. The printed materials may be used in isolation or in conjunction with the CD-ROMs, which offer scenarios, simulations, animations, and videos.

The first three supplements in the series (listed below) are designed for use in senior high school science classrooms:

- *Emerging and Re-emerging Infectious Diseases* (with expertise from the National Institute of Allergy and Infectious Diseases)
- *Cell Biology and Cancer* (with expertise from the National Cancer Institute)
- *Human Genetic Variation* (with expertise from the National Human Genome Research Institute)

We appreciate the invaluable contributions of the talented staff at Biological Sciences Curriculum Study (BSCS) and Videodiscovery, Inc., who developed these materials. We are also grateful to the scientific advisers at the NIH institutes who worked long and hard on this project. Finally, we thank the teachers and students across the country who participated in focus groups and field tests to help ensure that these materials are both engaging and effective.

We are eager to know about your particular experience with the supplements. Your comments help this program to evolve and grow. For continuing updates on the curriculum supplement series or to make comments, please visit http://science-education.nih.gov/supplements. You may also send your suggestions to

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I hope you find our series a valuable addition to your classroom and wish you a productive school year.

Bruce A. Fuchs, Ph.D.
Director
Office of Science Education
National Institutes of Health

1 The National Academy of Sciences released the *National Science Education Standards* in December 1995 to outline what all citizens should understand about science by the time they graduate from high school. The *Standards* encourage teachers to select major science concepts or themes that empower students to use information to solve problems rather than to stress memorization of large volumes of unconnected bits of information.

About the National Institutes of Health

The National Institutes of Health (NIH)—the world's top medical research center—is charged with addressing the health concerns of the nation. The NIH is the largest U.S. governmental sponsor of health studies conducted nationwide.

Simply described, the NIH's goal is to acquire new knowledge to help prevent, detect, diagnose, and treat disease and disability, from the rarest genetic disorder to the common cold. The NIH works toward that goal by conducting research in its own laboratories in Bethesda, Maryland; supporting the research of nonfederal scientists throughout the country and abroad; helping train research investigators; and fostering communication of medical information to the public.

The NIH Supports Research A principal concern of the NIH is to invest wisely the tax dollars entrusted to it for the support and conduct of medical research. Approximately 82 percent of the investment is made through grants and contracts supporting research and training in more than 2,000 universities, medical schools, hospitals, and research institutions throughout the United States and abroad.

Approximately 10 percent of the budget goes to more than 2,000 projects conducted mainly in NIH laboratories. About 8 percent covers support costs of research conducted both within and outside the NIH.

NIH Research Grants To apply for a research grant, an individual scientist must submit an idea in a written application. Each application undergoes a peer review process. A panel of scientific experts, who are active researchers in the medical sciences, first evaluates the scientific merit of the application. Then, a national advisory council or board, comprised of eminent scientists as well as public members who are interested in health issues or the medical sciences, determines the project's overall merit and priority. Because funds are limited, the process is very competitive.

The Nobelists The rosters of those who have conducted research, or who have received NIH support over the years, include some of the world's most illustrious scientists and physicians. Among them are 97 scientists who have won Nobel Prizes for achievements as diverse as deciphering the genetic code and learning what causes hepatitis.

Five Nobelists made their prize-winning discoveries in NIH laboratories: Doctors Christian B. Anfinsen, Julius Axelrod, D. Carleton Gajdusek, Marshall W. Nirenberg, and Martin Rodbell.

Impact of the NIH on the Nation's Health The research programs of the NIH have been remarkably successful during the past 50 years. NIH-funded scientists have made substantial progress in understanding the basic mechanisms of disease and have vastly improved the preventive, diagnostic, and therapeutic options available.

During the last few decades, NIH research played a major role in making possible achievements like these:

- Mortality from heart disease, the number one killer in the United States, dropped by 36 percent between 1977 and 1999.
- Improved treatments and detection methods increased the relative five-year survival rate for people with cancer to 60 percent.
- Those suffering from depression now look forward to returning to work and leisure activities, thanks to treatments that give them an 80 percent chance to resume a full life in a matter of weeks.
- Vaccines protect against infectious diseases that once killed and disabled millions of children and adults.
- In 1990, NIH researchers performed the first trial of gene therapy in humans. Scientists are increasingly able to locate, identify, and describe the functions of many of the genes in the human genome. The ultimate goal is to develop screening tools and gene therapies for the general population for cancer and many other diseases.

Educational and Training Opportunities at the NIH The NIH offers a myriad of opportunities including summer research positions for students. For details, visit http://science-education.nih.gov/students.

For more information about the NIH, visit http://www.nih.gov.

The NIH Office of Science Education

The NIH Office of Science Education (OSE) is bringing exciting new resources free of charge to science teachers of grades kindergarten through 12. OSE learning tools support teachers in training the next generation of scientists and scientifically literate citizens. These materials cover information not available in standard textbooks and allow students to explore biological concepts using real world examples. In addition to the curriculum supplement, OSE provides a host of valuable resources accessible through the OSE Web site (http://science-education.nih.gov), such as:

- **Snapshots of Science and Medicine.**[2] This online magazine—plus interactive learning tools—is designed for ease of use in high school science classrooms. Three issues, available for free, are published during the school year. Each focuses on a new area of research and includes four professionally written articles on findings, historical background, related ethical questions, and profiles of people working in the field. Also included are a teaching guide, classroom activities, handouts, and more. (http://science-education.nih.gov/snapshots)
- **Women Are Scientists Video and Poster Series.**[3] This series provides teachers and guidance counselors with free tools to encourage young women to pursue careers in the medical field. The informative, full-color video and poster sets focus on some of the careers in which women are currently underrepresented. The first set, titled "Women are Surgeons," has been completed. The second, "Women are Pathologists," will be finished in 2000, and the third, "Women are Researchers," in 2001. (http://science-education.nih.gov/women)
- **Internship Programs.** Visit the OSE Web site to obtain information on a variety of NIH programs open to teachers and students. (http://science-education.nih.gov/students)
- **National Science Teacher Conferences.** Thousands of copies of NIH materials are distributed to teachers for free at the OSE exhibit booth at conferences of the National Science Teachers Association and the National Association of Biology Teachers. OSE also offers teacher-training workshops at many conferences. (http://science-education.nih.gov/exhibits)

In the development of learning tools, OSE supports science education reform as outlined in the *National Science Education Standards* and related guidelines.

We welcome your comments about existing resources and suggestions about how we may best meet your needs. Feel free to send your comments to us at http://science-education.nih.gov/feedback.

2, 3 These projects are collaborative efforts between OSE and NIH Office of Research on Women's Health.

About the National Cancer Institute

The National Cancer Institute (NCI), a component of the NIH, is the federal government's principal agency for cancer research and training. The NCI coordinates the National Cancer Program, which conducts and supports research, training, health information dissemination, and other programs with respect to the cause, diagnosis, prevention and treatment of cancer, rehabilitation from cancer, and the continuing care of cancer patients and the families of cancer patients.

The NCI was established under the National Cancer Act of 1937. The National Cancer Act of 1971 broadened the scope and responsibilities of the NCI and created the National Cancer Program. Over the years, the NCI's mandate has come to include dissemination of current cancer information and assessment of the incorporation of state-of-the-art cancer treatments into clinical practice. Today, the NCI's activities include:

- supporting and coordinating research projects conducted by universities, hospitals, research foundations, and businesses throughout this country and abroad through research grants and cooperative agreements;
- conducting research in its own laboratories and clinics;
- supporting education and training in all areas of cancer research through training grants, fellowships, and "career awards" for longtime researchers;
- supporting a national network of Cancer Centers, which are hubs of cutting-edge research, high quality cancer care, and outreach and education for both health care professionals and the general public;
- collaborating with voluntary organizations and other national and foreign institutions engaged in cancer research and training activities;
- collaborating with partners in industry in a number of areas, including the development of technologies that are revolutionizing cancer research; and
- collecting and disseminating information about cancer.

For more information about the National Cancer Institute, visit its Web site at http://www.nci.nih.gov.

Introduction to the Module

"Tumors destroy man in a unique and appalling way, as flesh of his own flesh which has somehow been rendered proliferative, rampant, predatory, and ungovernable . . . Yet, despite more than 70 years of experimental study, they remain the least understood . . . What can be the why for these happenings?"

—Peyton Rous, in his acceptance lecture for the Nobel Prize in Physiology or Medicine (1966)

Late in 1910, a young scientist at Rockefeller University was preparing to conduct a most improbable experiment. He wanted to know if one chicken could "catch" cancer from another. At that time, the concept that every cell in the body is derived from another cell was new, and the idea that cancer might involve a disruption of normal cell growth was just taking hold. Thirty years had passed since Louis Pasteur's influential paper on germ theory dislodged the humoral theory of disease that had prevailed for more than 2,000 years, and the prevailing scientific view of cancer emphasized the role of chemical and physical agents, not infectious ones, as potential causes.

Nevertheless, the 30-year-old Peyton Rous was able to show that cell-free extracts from one chicken were able to cause the formation of the same type of tumor when injected into a second chicken. Rous' tumor extracts had been passed through a filter with pores so small that even bacteria were excluded. This result strongly implicated the newly-discovered "filterable agents" known as viruses. Rous was later able to demonstrate that other types of chicken tumors could also be spread by their own, unique "filterable agents," and that each would faithfully produce its original type of tumor (bone, cartilage, blood vessel) when injected into healthy animals.

Unfortunately, the full significance of these data was not to be realized for many decades. One reason was the difficulty of reproducing these results in mammals. But another reason was that scientists could not place Rous' discovery in a proper context. So many different things seemed to be associated with cancer that no one was able to make sense of it all. For example,

- In 1700, the Italian physician Bernardino Ramazzini wrote about the high rate of breast cancer among nuns and speculated that it was related to their celibacy and childlessness. This was the first indication that how one lived might affect the development of cancer.
- In 1775, Percivall Pott, a London physician, suggested that the very high rate of scrotal and nasal cancers among chimney sweeps was a result of their exposure to soot. This was the first indication that exposure to certain chemicals in the environment could be an important factor in cancer.
- In 1886, Hilario de Gouvea, a professor at the Medical School in Rio de Janeiro, reported the case of a family with an increased susceptibility to retinoblastoma, a form of cancer that normally occurs in only one out of about 20,000 children. This suggested that certain cancers have a hereditary basis.
- The discovery of x-rays in 1895 led to its association with the skin cancer on the hand of a lab technician by 1902. Within a decade, many more physicians and scientists, unaware of the dangers of radiation, developed a variety of cancers.
- In 1907, an epidemiological study found that the meat-eating Germans, Irish, and Scandinavians living in Chicago had higher rates of cancer than did Italians and Chinese who ate considerably less meat.

At the time Peyton Rous accepted his Nobel Prize, it was not clear how these, and many other observations would ever be reconciled. By the early

1970s, however, scientists armed with the new tools of molecular biology were about to revolutionize our understanding of cancer. In fact, just over three decades later, Rous would be astounded to learn of the progress made answering his question of "why."

Cell Biology and Cancer has two objectives. The first objective is to introduce students to major concepts related to the development and impact of cancer. Today we have a picture of cancer that, while still incomplete, is remarkably coherent and precise. Cancer develops when mutations occur in genes that normally operate to control cell division. These mutations prompt the cell to divide inappropriately. Cancer-causing mutations can be induced by a wide variety of environmental agents and even several known viruses. Such mutations also can be inherited—thus, the observation that some families have a higher risk for developing cancer than others. We still have much to learn about cancer, to be sure, but the clarity and detail of our understanding today speak powerfully of the enormous gains scientists have made in just the last 30 years. One objective of this module is to help students catch a bit of the excitement of these gains.

A second objective is to convey to students the relationship between basic biomedical research and the improvement of personal and public health. Cancer-related research has yielded many benefits for humankind. Most directly, it has guided the development of public health policies and medical interventions that today are helping us prevent, treat, and often, even cure cancer. A dramatic illustration of the success that scientists and health care specialists are having in the war against cancer came in the 1998 announcement by the National Cancer Institute, the American Cancer Society, and the Centers for Disease Control and Prevention that cancer incidence and death rates for all cancers combined and for most of the top 10 sites declined between 1990 and 1995, reversing an almost 20-year trend of increasing cancer cases and death rates in the United States.

Research is also pointing the way to new therapies, therapies that scientists hope will combat the disease without as many of the devastating side

Figure 1 For people touched by cancer, modern science offers better treatment and brighter prospects than ever before.

effects of current treatments. For example, the development of drugs that target the genes, proteins, and pathways unique to cancer cells represents a radical leap forward in cancer treatment. Although most of these drugs are only beginning to be tested, preliminary results offer reason for enthusiasm about the prospects of controlling cancer at its molecular level.

And cancer research has yielded other benefits as well. In particular, it has vastly improved our understanding of many of the body's most critical cellular and molecular processes. The need to understand cancer has spurred research into the normal cell cycle, mutation, DNA repair, growth factors, cell signaling, and cell aging and death. Research also has led to an improved understanding of cell adhesion and anchorage, the "address" system that keeps normal cells from establishing themselves in inappropriate parts of the body, angiogenesis (the formation of blood vessels), and the role of the immune system in protecting the body from harm from within as well as without.

This module addresses our progress in understanding the cellular and molecular basis of cancer and considers the impact of what we have learned for individuals and society. There are many concepts we could have addressed, but we have chosen, with the help of a wide variety of experts in this field, a relatively small number for exploration by your students. Those concepts follow.

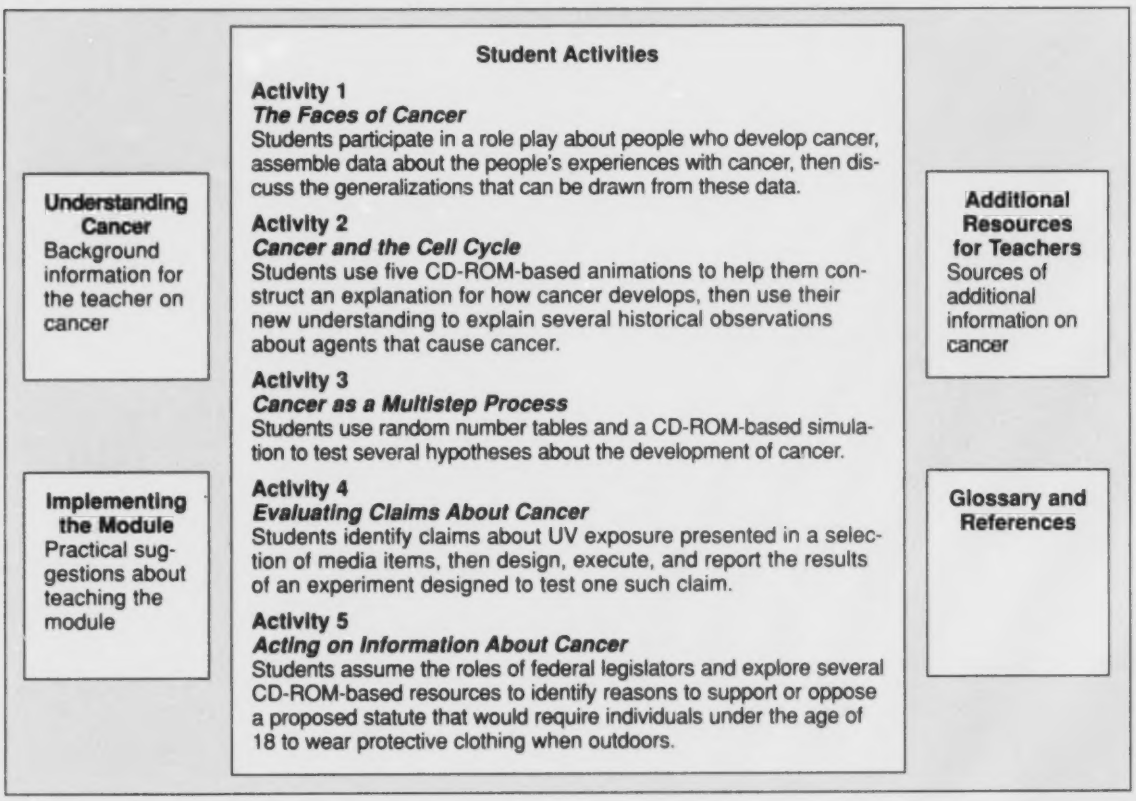
- Cancer is a group of more than 100 diseases that develop across time. Cancer can develop

in virtually any of the body's tissues, and both hereditary and environmental factors contribute to its development.

- The growth and differentiation of cells in the body normally are precisely regulated; this regulation is fundamental to the orderly process of development that we observe across the life spans of multicellular organisms. Cancer develops due to the loss of growth control in cells. Loss of control occurs as a result of mutations in genes that are involved in cell cycle control.
- No single event is enough to turn a cell into a cancerous cell. Instead, it seems that the accumulation of damage to a number of genes ("multiple hits") across time leads to cancer.
- Scientists use systematic and rigorous criteria to evaluate claims about factors associated with cancer. Consumers can evaluate such claims by applying criteria related to the source, certainty, and reasonableness of the supporting information.
- We can use our understanding of the science of cancer to improve personal and public health. Translating our understanding of science into public policy can raise a variety of issues, such as the degree to which society should govern the health practices of individuals. Such issues often involve a tension between the values of preserving personal and public health and preserving individual freedom and autonomy.

We hope that the five activities provided in this module (Figure 2) will be effective vehicles to carry these concepts to your students. Although the activities contain much interesting information about various types of cancer, we suggest that you focus your students' attention on the major concepts the module was designed to convey. The concluding steps in each activity are intended to remind students of those concepts as the activity draws to a close.

Figure 2 This diagram identifies the module's major sections and describes their contents.


Understanding Cancer
Background information for the teacher on cancer
Implementing the Module
Practical suggestions about teaching the module
Student Activities
Activity 1
The Faces of Cancer
Students participate in a role play about people who develop cancer, assemble data about the people's experiences with cancer, then discuss the generalizations that can be drawn from these data.
Activity 2
Cancer and the Cell Cycle
Students use five CD-ROM-based animations to help them construct an explanation for how cancer develops, then use their new understanding to explain several historical observations about agents that cause cancer.
Activity 3
Cancer as a Multistep Process
Students use random number tables and a CD-ROM-based simulation to test several hypotheses about the development of cancer.
Activity 4
Evaluating Claims About Cancer
Students identify claims about UV exposure presented in a selection of media items, then design, execute, and report the results of an experiment designed to test one such claim.
Activity 5
Acting on Information About Cancer
Students assume the roles of federal legislators and explore several CD-ROM-based resources to identify reasons to support or oppose a proposed statute that would require individuals under the age of 18 to wear protective clothing when outdoors.
Additional Resources for Teachers
Sources of additional information on cancer
Glossary and References

Understanding Cancer

In simple terms, **cancer** is a group of more than 100 diseases that develop across time and involve the uncontrolled division of the body's cells. Although cancer can develop in virtually any of the body's tissues, and each type of cancer has its unique features, the basic processes that produce cancer are quite similar in all forms of the disease.

Cancer begins when a cell breaks free from the normal restraints on cell division and begins to follow its own agenda for proliferation (Figure 3). All of the cells produced by division of this first, ancestral cell and its progeny also display inappropriate proliferation. A **tumor**, or mass of cells, formed of these abnormal cells may remain within the tissue in which it originated (a condition called in situ cancer), or it may begin to invade nearby tissues (a condition called invasive cancer). An invasive tumor is said to be **malignant**, and cells shed into the blood or lymph from a malignant tumor are likely to establish new tumors (**metastases**) throughout the body. Tumors threaten an individual's life when their growth disrupts the tissues and organs needed for survival.

What happens to cause a cell to become cancerous? Thirty years ago, scientists could not offer a coherent answer to this question. They knew that

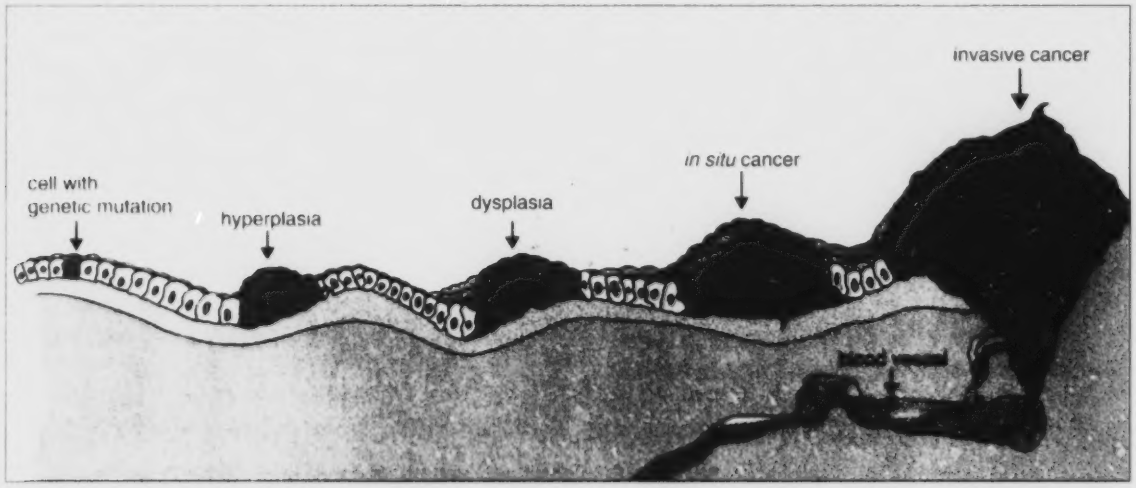


Figure 3 The stages of tumor development. A malignant tumor develops across time, as shown in this diagram. This tumor develops as a result of four mutations, but the number of mutations involved in other types of tumors can vary. We do not know the exact number of mutations required for a normal cell to become a fully malignant cell, but the number is probably less than ten. **a.** The tumor begins to develop when a cell experiences a mutation that makes the cell more likely to divide than it normally would. **b.** The altered cell and its descendants grow and divide too often, a condition called hyperplasia. At some point, one of these cells experiences another mutation that further increases its tendency to divide. **c.** This cell's descendants divide excessively and look abnormal, a condition called dysplasia. As time passes, one of the cells experiences yet another mutation. **d.** This cell and its descendants are very abnormal in both growth and appearance. If the tumor that has formed from these cells is still contained within its tissue of origin, it is called in situ cancer. In situ cancer may remain contained indefinitely. **e.** If some cells experience additional mutations that allow the tumor to invade neighboring tissues and shed cells into the blood or lymph, the tumor is said to be malignant. The escaped cells may establish new tumors (metastases) at other locations in the body.

cancer arose from cells that began to proliferate uncontrollably within the body, and they knew that chemicals, radiation, and viruses could trigger this change. But exactly how it happened was a mystery.

Research across the last three decades, however, has revolutionized our understanding of cancer. In large part, this success was made possible by the development and application of the techniques of molecular biology, techniques that enabled researchers to probe and describe features of individual cells in ways unimaginable a century ago. Today, we know that cancer is a disease of molecules and genes, and we even know many of the molecules and genes involved. In fact, our increasing understanding of these genes is making possible the development of exciting new strategies for avoiding, forestalling, and even correcting the changes that lead to cancer.

Unraveling the Mystery of Cancer

People likely have wondered about the cause of cancer for centuries. Its name derives from an observation by Hippocrates more than 2,300 years ago that the long, distended veins that radiate out from some breast tumors look like the limbs of a crab. From that observation came the term *karkinoma* in Greek, and later, *cancer* in Latin.

With the work of Hooke in the 1600s, and then Virchow in the 1800s, came the understanding that living tissues are composed of cells, and that all cells arise as direct descendants of other cells. Yet, this understanding raised more questions about cancer than it answered. Now scientists began to ask from what kinds of normal cells cancer cells arise, how cancer cells differ from their normal counterparts, and what events promote the proliferation of these abnormal cells. And physicians began to ask how cancer could be prevented or cured.

Clues from epidemiology. One of the most important early observations that people made about cancer was that its incidence varies between different populations. For example, in 1775, an extraordinarily high incidence of scrotal cancer was described among men who worked as chimney sweeps as boys. In the mid-1800s, lung cancer was observed at alarmingly high rates among pitchblende miners in Germany. And by the end of the 19th century, using snuff and cigars was thought by some physicians to be closely associated with cancers of the mouth and throat.

These observations and others suggested that the origin or causes of cancer may lie outside the body and, more important, that cancer could be linked to identifiable and even preventable causes. These ideas led to a widespread search for agents that might cause cancer. One early notion, prompted by the discovery that bacteria cause a variety of important human diseases, was that cancer is an infectious disease. Another idea was that cancer arises from the chronic irritation of tissues. This view received strong support with the discovery of X-rays in 1895 and the observation that exposure to this form of radiation could induce localized tissue damage, which could lead in turn to the development of cancer. A conflicting view, prompted by the observation that cancer sometimes seems to run in families, was that cancer is hereditary.

Such explanations, based as they were on fragmentary evidence and incomplete understanding, helped create the very considerable confusion about cancer that existed among scientists well into the mid-twentieth century. The obvious question facing researchers—and no one could seem to answer it—was how agents as diverse as this could all cause cancer. Far from bringing science closer to understanding cancer, each new observation seemed to add to the confusion.

Yet each new observation also, ultimately, contributed to scientists' eventual understanding of the disease. For example, the discovery in 1910 that a defined, submicroscopic agent isolated from a chicken tumor could induce new tumors in healthy chickens showed that a tumor could be traced simply and definitively back to a single cause. Today, scientists know this agent as Rous sarcoma virus, one of several viruses that can act as causative factors in the development of cancer.

Although cancer-causing viruses are not prime agents in promoting most human cancers, their intensive study focused researchers' attention on cellular genes as playing a central role in the development of the disease.

Likewise, investigations into the association between cancer and tissue damage, particularly that induced by radiation, revealed that while visible damage sometimes occurs, something more subtle happens in cells exposed to cancer-causing agents. One clue to what happens came from the work of Herman Muller, who noticed in 1927 that X-irradiation of fruit flies often resulted in mutant offspring. Might the two known effects of X-rays, promotion of cancer and genetic mutation, be related to one another? And might chemical carcinogens induce cancer through a similar ability to damage genes?

Support for this idea came from the work of Bruce Ames and others who showed in 1975 that compounds known to be potent carcinogens (cancer-causing agents) generally also were potent mutagens (mutation-inducing agents), and that compounds known to be only weak carcinogens were only weak mutagens. Although scientists know today that many chemicals do not follow this correlation precisely, this initial, dramatic association between mutagenicity and carcinogenicity had widespread influence on the development of a unified view of the origin and development of cancer.

Finally, a simple genetic model, proposed by Alfred Knudson in 1971, provided both a compelling explanation for the origins of retinoblastoma, a rare tumor that occurs early in life, and a convincing way to reconcile the view of cancer as a disease produced by external agents that damage cells with the observation that some cancers run in families. Knudson's model states that children with sporadic retinoblastoma (children whose parents have no history of the disease) are genetically normal at the moment of conception, but experience two somatic mutations that lead to the development of an eye tumor. Children with familial retinoblastoma (children whose parents have a history of the disease) already carry one mutation at conception and thus must experience only one more mutation to reach the doubly mutated configuration required for a tumor to form. In effect, in familial retinoblastoma, each retinal cell is already primed for tumor development, needing only a second mutational event to trigger the cancerous state. The difference in probabilities between the requirement for one or two mutational events, happening randomly, explains why in sporadic retinoblastoma, the affected children have only one tumor focus, in one eye, while in familial retinoblastoma, the affected children usually have multiple tumor foci growing in both eyes.

Although it was years before Knudson's explanation was confirmed, it had great impact on scientists' understanding of cancer. Retinoblastoma, and by extension, other familial tumors, appeared to be linked to the inheritance of mutated versions of growth-suppressing genes. This idea led to the notion that cells in sporadically arising tumors might also have experienced damage to these critical genes as the cells moved along the path from the normal to the cancerous state.

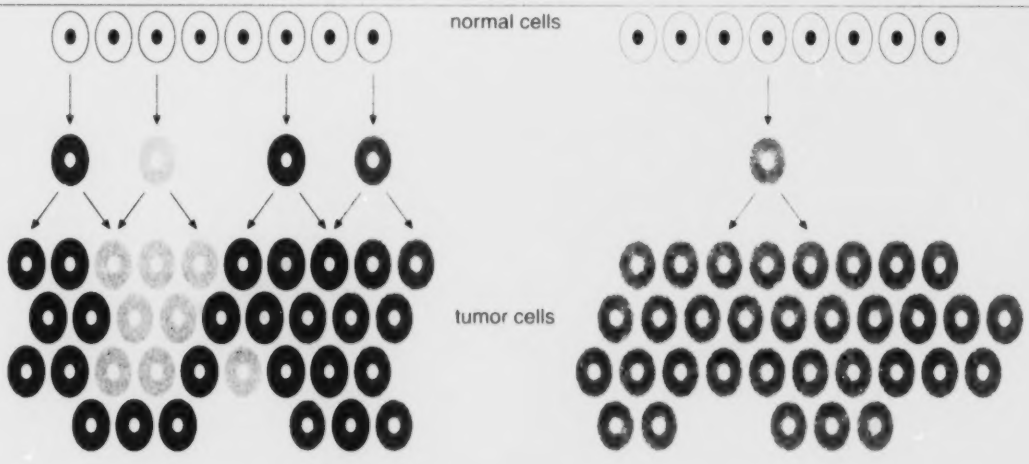
Clues from cell biology. Another field of study that contributed to scientists' growing understanding of cancer was cell biology. Cell biologists studied the characteristics of cancer cells, through observations in the laboratory and by inferences from their appearance in the whole organism. Not unexpectedly, these investigations yielded a wealth of information about normal cellular processes. But they also led to several key understandings about cancer, understandings that ultimately allowed scientists to construct a unified view of the disease.

One such understanding is that cancer cells are indigenous cells—abnormal cells that arise from the body's normal tissues. Furthermore, virtually all malignant tumors are monoclonal in origin, that is, derived from a single ancestral cell that somehow underwent conversion from a normal to a cancerous state. These insights, as straightforward as they seem, were surprisingly difficult to reach. How could biologists describe the cell pedigree of a mass of cells that eventually is recognized as a tumor?

One approach to identifying the origin of cancer cells came from attempts to transplant tissues from one person to another. Such transplants work well between identical twins, but less well as the people involved are more distantly related. The barrier to successful transplantation exists because the recipient's immune system can distinguish between cells that have always lived inside the self and cells of foreign origin. One practical application of this discovery is that tissues can be classified as matching or nonmatching before a doctor attempts to graft a tissue or organ into another person's body. Such tissue-typing tests, when done on cancer cells, reveal that the tumor cells of a particular cancer patient are always of the same transplantation type as the cells of normal tissues located elsewhere in the person's body. Tumors, therefore, arise from one's own tissues, not from cells introduced into the body by infection from another person.

How do we know that tumors are monoclonal? Two distinct scenarios might explain how cancers develop within normal tissues. In the first, many individual cells become cancerous, and the resulting tumor represents the descendants of these original cells. In this case, the tumor is polyclonal in nature (Figure 4). In the second scenario, only one cell experiences the original transformation from a normal cell to a cancerous cell, and all of the cells in the tumor are descendants of that cell.

Direct evidence supporting the monoclonal origin of virtually all malignant tumors has been difficult to acquire because most tumor cells lack obvious distinguishing marks that scientists can use to demonstrate their clonal relationship. There is, however, one cellular marker that scientists can use as an indication of such relationships: the inactivated X chromosome that occurs in almost all of the body cells of a human female. X-chromosome inactivation occurs randomly in all cells during female embryonic development. Because the inactivation is random, the female is like a mosaic in terms of the X chromosome, with different copies of the X turned on or off in different cells of the body. Once inactivation occurs in a cell, all of the future generations of cells coming from that cell have the same chromosome inactivated in them as well (either the maternal or the paternal X). The observation that all the cells within a given tumor invariably have the same X chromosome inactivated suggests that all cells in the tumor must have descended from a single ancestral cell.



Source: Modified from Varmus, A., & Weinberg, R.A. 1993. *Genes and the biology of cancer.* New York: Scientific American Library.

Figure 4 Two schemes by which tumors can develop. Most—if not all—human cancer appears to be monoclonal.

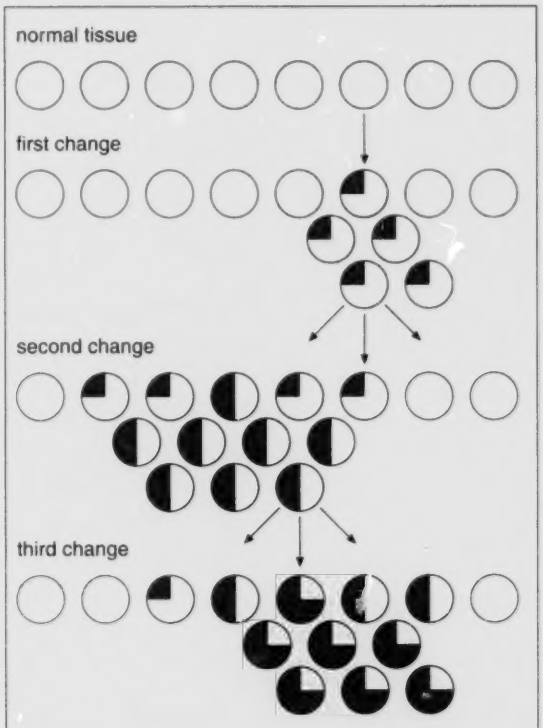
Cancer, then, is a disease in which a single normal body cell undergoes a genetic transformation into a cancer cell. This cell and its descendants, proliferating across many years, produce the population of cells that we recognize as a tumor, and tumors produce the symptoms that an individual experiences as cancer.

Even this picture, although accurate in its essence, did not represent a complete description of the events involved in tumor formation. Additional research revealed that as a tumor develops, the cells of which it is composed become different from one another as they acquire new traits and form distinct subpopulations of cells within the tumor. As shown in Figure 5, these changes allow the cells that experience them to compete with increasing success against cells that lack the full set of changes. The development of cancer, then, occurs as a result of a series of clonal expansions from a single ancestral cell.

A second critical understanding that emerged from studying the biology of cancer cells is that these cells show a wide range of important differences from normal cells. For example, cancer cells are genetically unstable and prone to rearrangements, duplications, and deletions of their chromosomes that cause their progeny to display unusual traits. Thus, although a tumor as a whole is monoclonal in origin, it may contain a large number of cells with diverse characteristics.

Cancerous cells also look and act differently from normal cells. In most normal cells, the nucleus is only about one-fifth the size of the cell; in cancerous cells, the nucleus may occupy most of the cell's volume. Tumor cells also often lack the differentiated traits of the normal cell from which they arose. Whereas normal secretory cells produce and release mucus, cancers derived from these cells may have lost this characteristic. Likewise, epithelial cells usually contain large amounts of keratin, but the cells that make up skin cancer may no longer accumulate this protein in their cytoplasms.

The key difference between normal and cancerous cells, however, is that cancer cells have lost the restraints on growth that characterize normal cells. Significantly, a large number of cells in a tumor are engaged in mitosis, whereas mitosis is a relatively rare event in most normal tissues. Cancer cells also demonstrate a variety of unusual characteristics when grown in culture; two such examples are a lack of contact inhibition and a reduced dependence on the presence of growth factors in the environment. In contrast to normal cells, cancer cells do not cooperate with other cells in their environment. They often proliferate indefinitely in tissue culture. The ability to divide for an apparently unlimited number of generations is another important characteristic of the cancerous state, allowing a tumor composed of such cells to grow



Source: Modified from Varmus, A., & Weinberg, R.A. 1993. *Genes and the biology of cancer.* New York: Scientific American Library.

Figure 5 A series of changes leads to tumor formation. Tumor formation occurs as a result of successive clonal expansions. This figure illustrates only three such changes; the development of many cancers likely involves more than three.

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without the constraints that normally limit cell growth.

A unified view. By the mid-1970s, scientists had started to develop the basis of our modern molecular understanding of cancer. In particular, the relationship Ames and others had established between mutagenicity and carcinogenicity provided substantial support for the idea that chemical carcinogens act directly through their ability to damage cellular genes. This idea led to a straightforward model for the initiation of cancer: Carcinogens induce mutations in critical genes, and these mutations direct the cell in which they occur, as well as all of its progeny cells, to grow abnormally. The result of this abnormal growth appears years later as a tumor. The model could even explain the observation that cancer sometimes appears to run in families: If cancer is caused by mutations in critical genes, then people who inherit such mutations would be more susceptible to cancer's development than people who do not.

As exciting as it was to see a unified view of cancer begin to emerge from the earlier confusion, cancer researchers knew their work was not finished. The primary flaw in their emerging explanation was that the nature of these cancer-causing mutations was unknown. Indeed, their very existence had yet to be proven. Evidence from work with cancer-causing viruses suggested that only a small number of genes were involved, and evidence from cell biology pointed to genes that normally control cell division. But now scientists asked new questions: Exactly which genes are involved? What are their specific roles in the cell? and How do their functions change as a result of mutation?

It would take another 20 years and a revolution in the techniques of biological research to answer these questions. However, today our picture of the causes and development of cancer is so detailed that scientists find themselves in the extraordinary position of not only knowing many of the genes involved but also being able to target prevention, detection, and treatment efforts directly at these genes.

Cancer as a Multistep Process

A central feature of today's molecular view of cancer is that cancer does not develop all at once, but across time, as a long and complex succession of genetic changes. Each change enables precancerous cells to acquire some of the traits that together create the malignant growth of cancer cells.

Two categories of genes play major roles in triggering cancer. In their normal forms, these genes control the **cell cycle**, the sequence of events by which cells enlarge and divide. One category of genes, called **proto-oncogenes**, encourages cell division. The other category, called **tumor suppressor genes**, inhibits it. Together, proto-oncogenes and tumor suppressor genes coordinate the regulated growth that normally ensures that each tissue and organ in the body maintains a size and structure that meets the body's needs.

What happens when proto-oncogenes or tumor suppressor genes are mutated? Mutated proto-oncogenes become oncogenes, genes that stimulate excessive division. And mutations in tumor suppressor genes inactivate these genes, eliminating the critical inhibition of cell division that normally prevents excessive growth. Collectively, mutations in these two categories of genes account for much of the uncontrolled cell division that occurs in human cancers (Figure 6).

The role of oncogenes. How do proto-oncogenes, or more accurately, the oncogenes they become after mutation, contribute to the development of cancer? Most proto-oncogenes code for proteins that are involved in molecular pathways that receive and process growth-stimulating signals from other cells in a tissue. Typically, such signaling begins with the production of a growth factor, a protein that stimulates division. These growth factors move through the spaces between cells and attach to specific receptor proteins located on the surfaces of neighboring cells. When a growth-stimulating factor binds to such a receptor, the receptor conveys a stimulatory signal to proteins in the cytoplasm. These proteins emit stimulatory signals to other proteins in the cell until the division-promoting

Oncogenes

PDGF	codes for a protein called platelet-derived growth factor (involved in some forms of brain cancer)
Ki-ras	codes for a protein involved in a stimulatory signaling pathway (involved in lung, ovarian, colon, and pancreatic cancer)
MDM2	codes for a protein that is an antagonist of the *p53* tumor suppressor protein (involved in certain connective tissue cancers)

Tumor Suppressor Genes

NF-1	codes for a protein that inhibits a stimulatory protein (involved in myeloid leukemia)
RB	codes for the pRB protein, a key inhibitor of the cell cycle (involved in retinoblastoma and bone, bladder, and breast cancer)
BRCA1	codes for a protein whose function is still unknown (involved in breast and ovarian cancers)

Figure 6 Some Genes Involved in Human Cancer

message reaches the cell's nucleus and activates a set of genes that help move the cell through its growth cycle.

Oncogenes, the mutated forms of these proto-oncogenes, cause the proteins involved in these growth-promoting pathways to be overactive. Thus, the cell proliferates much faster than it would if the mutation had not occurred. Some oncogenes cause cells to overproduce growth factors. These factors stimulate the growth of neighboring cells, but they also may drive excessive division of the cells that just produced them. Other oncogenes produce aberrant receptor proteins that release stimulatory signals into the cytoplasm even when no growth factors are present in the environment. Still other oncogenes disrupt parts of the signal cascade that occurs in a cell's cytoplasm such that the cell's nucleus receives stimulatory messages continuously, even when growth factor receptors are not prompting them.

The role of tumor suppressor genes. To become cancerous, cells also must break free from the inhibitory messages that normally counterbalance these growth-stimulating pathways. In normal cells, inhibitory messages flow to a cell's nucleus much like stimulatory messages do. But when this flow is interrupted, the cell can ignore the normally powerful inhibitory messages at its surface.

Scientists are still trying to identify the normal functions of many known tumor suppressor genes. Some of these genes apparently code for proteins that operate as parts of specific inhibitory pathways. When a mutation causes such proteins to be inactivate or absent, these inhibitory pathways no longer function normally. Other tumor suppressor genes appear to block the flow of signals through growth-stimulating pathways; when these genes no longer function properly, such growth-promoting pathways may operate without normal restraint. Mutations in all tumor suppressor genes, however, apparently inactivate critical tumor suppressor proteins, depriving cells of this restraint on cell division.

The body's back-up systems. In addition to the controls on proliferation afforded by the coordinated action of proto-oncogenes and tumor suppressor genes, cells also have at least three other systems that can help them avoid runaway cell division. The first of these systems is the DNA repair system. This system operates in virtually every cell in the body, detecting and correcting errors in DNA. Across a lifetime, a person's genes are under constant attack, both by carcinogens imported from the environment and by chemicals produced in the cell itself. Errors also occur during DNA replication. In most cases, such errors are rapidly corrected by the cell's DNA repair system. Should the system fail, however, the error (now a mutation) becomes a permanent feature in that cell and in all of its descendants.

The system's normally high efficiency is one reason why many years typically must pass before all the mutations required for cancer to develop occur together in one cell. Mutations in DNA repair genes themselves, however, can undermine this repair system in a particularly devastating way: They damage a cell's ability to repair errors in its DNA. As a result, mutations appear in the cell (including mutations in genes that control cell growth) much more frequently than normal.

A second cellular back-up system prompts a cell to commit suicide (undergo **apoptosis**) if some essential component is damaged or its control system is deregulated. This observation suggests that tumors arise from cells that have managed to evade such death. One way of avoiding apoptosis involves the p53 protein. In its normal form, this protein not only halts cell division, but induces apoptosis in abnormal cells. The product of a tumor suppressor gene, p53 is inactivated in many types of cancers.

This ability to avoid apoptosis endangers cancer patients in two ways. First, it contributes to the growth of tumors. Second, it makes cancer cells resistant to treatment. Scientists used to think that radiation and chemotherapeutic drugs killed cancer cells directly by harming their DNA. It seems clear now that such therapy only slightly damages the DNA in cells; the damaged cells, in response, actively kill themselves. This discovery suggests that cancer cells able to evade apoptosis will be less responsive to treatment than other cells.

A third back-up system limits the number of times a cell can divide, and so assures that cells cannot reproduce endlessly. This system is governed by a counting mechanism that involves the DNA segments at the ends of chromosomes. Called telomeres, these segments shorten each time a chromosome replicates. Once the telomeres are shorter than some threshold length, they trigger an internal signal that causes the cell to stop dividing. If the cells continue dividing, further shortening of the telomeres eventually causes the chromosomes to break apart or fuse with one another, a genetic crisis that is inevitably fatal to the cell.

Early observations of cancer cells grown in culture revealed that, unlike normal cells, cancer cells can proliferate indefinitely. Scientists have recently discovered the molecular basis for this characteristic—an enzyme called telomerase, that systematically replaces telomeric segments that are trimmed away during each round of cell division. Telomerase is virtually absent from most mature cells, but is present in most cancer cells, where its action enables the cells to proliferate endlessly.

The multistep development of cancer. Cancer, then, does not develop all at once as a massive shift in cellular functions that results from a mutation in one or two wayward genes. Instead, it develops step-by-step, across time, as an accumulation of many molecular changes, each contributing some of the characteristics that eventually produce the malignant state. The number of cell divisions that occur during this process can be astronomically large—human tumors often become apparent only after they have grown to a size of 10 billion to 100 billion cells. As you might expect, the time frame involved also is very long—it normally takes decades to accumulate enough mutations to reach a malignant state.

Understanding cancer as a multistep process that occurs across long periods of time explains a number of long-standing observations. A key observation is the increase in incidence with age. Cancer is, for the most part, a disease of people who have lived long enough to have experienced a complex and extended succession of events. Because each change is a rare accident requiring years to occur, the whole process takes a very long time, and most of us die from other causes before it is complete.

Understanding cancer in this way also explains the increase in cancer incidence in people who experience unusual exposure to carcinogens, as well as the increased cancer risk of people who inherit predisposing mutations. Exposure to carcinogens increases the likelihood that certain harmful changes will occur, greatly increasing the probability of developing cancer during a normal life span. Similarly, inheriting a cancer-susceptibility mutation means that instead of that mutation being a rare event, it already has occurred, and not just in one or two cells, but in all the body's cells. In other words, the process of tumor formation has leapfrogged over one of its early steps. Now the accumulation of changes required to reach the malignant state, which usually requires several decades to occur, may take place in one or two.

Finally, understanding the development of cancer as a multistep process also explains the lag time that often separates exposure to a cancer-causing

agent and the development of cancer. This explains, for example, the observation that severe sunburns in children can lead to the development of skin cancer decades later. It also explains the 20- to 25-year lag between the onset of widespread cigarette smoking among women after World War II and the massive increase in lung cancer that occurred among women in the 1970s.

The Human Face of Cancer

For most Americans, the real issues associated with cancer are personal. More than 8 million Americans alive today have a history of cancer (National Cancer Institute, 1998; Rennie, 1996). In fact, cancer is the second leading cause of death in the United States, exceeded only by heart disease.

Who are these people who develop cancer and what are their chances for surviving it? Scientists measure the impact of cancer in a population by looking at a combination of three elements: (1) the number of new cases per year per 100,000 persons (**incidence rate**), (2) the number of deaths per 100,000 persons per year (**mortality rate**), and (3) the proportion of patients alive at some point after their diagnosis of cancer (**survival rate**). Data on incidence, mortality, and survival are collected from a variety of sources. For example, in the United States there are many statewide cancer registries and some regional registries based on groups of counties, many of which surround large metropolitan areas. Some of these population-based registries keep track of cancer incidence in their geographic areas only; others also collect follow-up information to calculate survival rates.

In 1973, the National Cancer Institute began the Surveillance, Epidemiology, and End Results (SEER) Program to estimate cancer incidence and patient survival in the United States. SEER collects cancer incidence data in 11 geographic areas and two supplemental registries, for a combined population of approximately 14 percent of the entire U.S. population. Data from SEER are used to track cancer incidence in the United States by primary cancer site, race, sex, age, and year of diagnosis. For example, Figure 7 shows SEER data for the age-adjusted cancer incidence rates for the 10 most common sites for Caucasian and African-American males and females for the period 1987-1991.

Cancer among children is relatively rare. SEER data from 1991 showed an incidence of only 14.1 cases per 100,000 children under age 15. Nevertheless, after accidents, cancer is the second leading cause of childhood death in the United States. Leukemias (4.3 per 100,000) and cancer of the brain and other nervous system organs (3.4 per 100,000) account for more than one-half of the cancers among children.

Everyone is at some risk of developing cancer. Cancer researchers use the term **lifetime risk** to indicate the probability that a person will develop cancer over the course of a lifetime. In the United States, men have a 1 in 2 lifetime risk of developing cancer, and women have a 1 in 3 risk.

For a specific individual, however, the risk of developing a particular type of cancer may be quite different from his or her lifetime risk of developing any type of cancer. **Relative risk** compares the risk of developing cancer between persons with a certain exposure or characteristic and persons who do not have this exposure or characteristic. For example, a person who smokes has a 10- to 20-fold higher relative risk of developing lung cancer compared with a person who does not smoke. This means that a smoker is 10- to 20-times more likely to develop lung cancer than a nonsmoker.

Scientists rely heavily on epidemiology to help them identify factors associated with the development of cancer. Epidemiologists look for factors that are common to cancer victims' histories and lives and evaluate these factors in the light of current understandings of the disease. With enough study, researchers may assemble evidence that a particular factor "causes" cancer, that is, that exposure to it increases significantly the probability of the disease developing. Although this information cannot be used to predict what will happen to any one individual exposed to this risk factor, it can help people make choices that reduce their exposure to known **carcinogens** (cancer-causing agents) and increase the probability that if cancer develops, it will be detected early (for

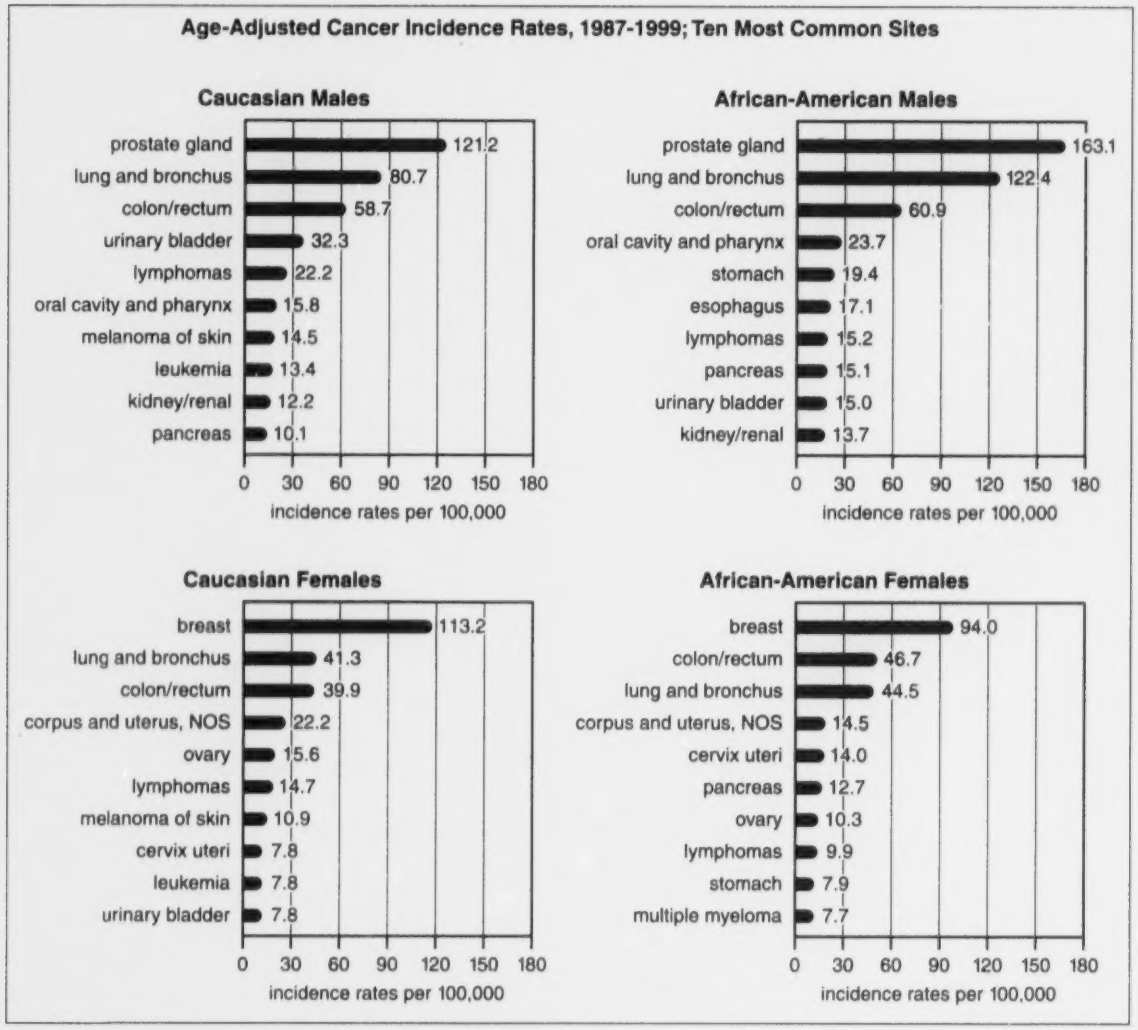

Figure 7 Age-Adjusted Cancer Incidence Rates, 1987-1991

example, by getting regular check-ups and participating in cancer screening programs).

As noted above, hereditary factors also can contribute to the development of cancer. Some people are born with mutations that directly promote the unrestrained growth of certain cells or the occurrence of more mutations. These mutations, such as the mutation identified in the 1980s that causes retinoblastoma, confer a high relative cancer risk. Such mutations are rare in the population, however, accounting for the development of fewer than 5 percent of the cases of fatal cancer.

Hereditary factors also contribute to the development of cancer by dictating a person's general physiological traits. For example, a person with fair skin is more susceptible to the development of skin cancer than a person with a darker complexion. Likewise, a person whose body metabolizes and eliminates a particular carcinogen relatively inefficiently is more likely to develop types of cancer associated with that carcinogen than a person who has more efficient forms of the genes involved in that particular metabolic process. These inherited characteristics do not directly promote the

development of cancer; each person, susceptible or not, still must be exposed to the related environmental carcinogen for cancer to develop. Nevertheless, genes probably do contribute in some way to the vast majority of cancers.

One question often asked about cancer is "How many cases of cancer would be expected to occur naturally in a population of individuals who somehow had managed to avoid all environmental carcinogens and also had no mutations that predisposed them to developing cancer?" Comparing populations around the world with very different cancer patterns has led epidemiologists to suggest that perhaps only about 25 percent of all cancers are "hard core"—that is, would develop anyway, even in a world free of external influences. These cancers would occur simply because of the production of carcinogens within the body and because of the random occurrence of unrepaired genetic mistakes.

Although cancer continues to be a significant health issue in the United States, a recent report from the American Cancer Society (ACS), National Cancer Institute (NCI), and Centers for Disease Control and Prevention (CDC) indicates that health officials are making progress in controlling the disease. In a news bulletin released on 12 March 1998, the ACS, NCI, and CDC announced the first sustained decline in the cancer death rate, a turning point from the steady increase observed throughout much of the century. The report showed that after increasing 1.2 percent per year from 1973 to 1990, the incidence for all cancers combined declined an average of 0.7 percent per year from 1990 to 1995. The overall cancer death rate also declined by about 0.5 percent per year across this period.

The overall survival rate for all cancer sites combined also continues to increase steadily, from 49.3 percent in 1974–1976 to 53.9 percent in 1983–1990 (Figure 8). In some cases—for example, among children age 15 and younger—survival rates have increased dramatically.

New Hope for Treating Cancer

What explanation can we offer for the steady increase in survival rates among cancer patients? One answer likely is the improvements scientists have made in cancer detection.

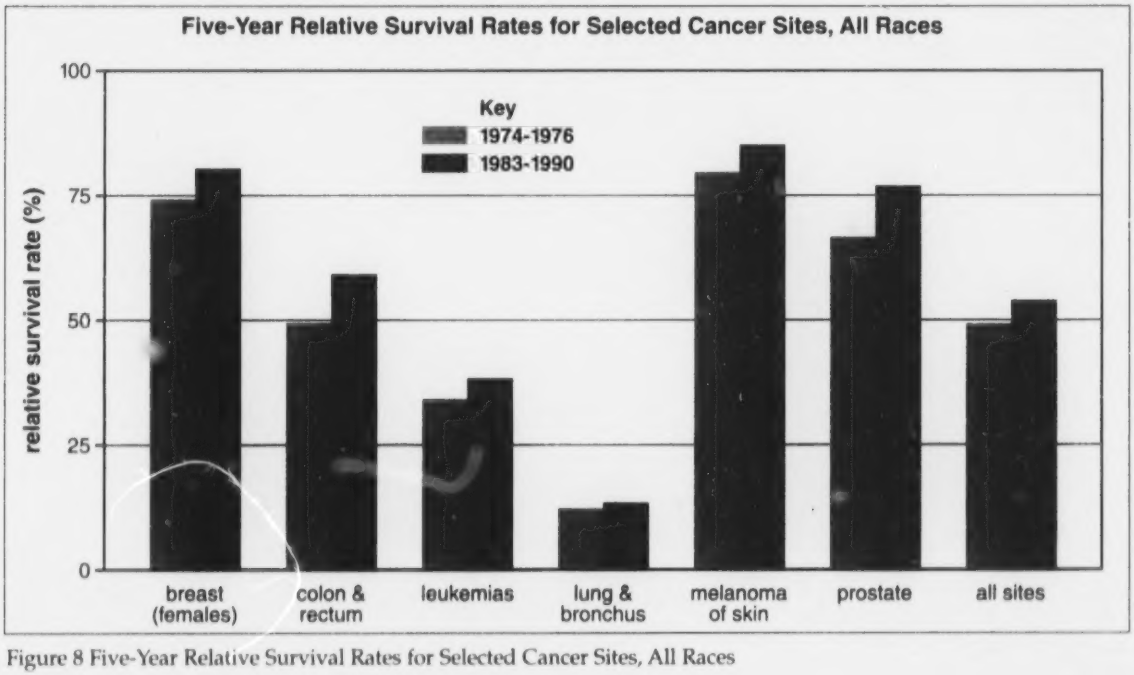

Figure 8 Five-Year Relative Survival Rates for Selected Cancer Sites, All Races

These improvements include a variety of new imaging techniques as well as blood and other tests that can help physicians detect and diagnose cancer early. Although many Americans regularly watch for the early symptoms of cancer, by the time symptoms occur many tumors already have grown quite large and may have metastasized. Likewise, many cancers have no symptoms. Clearly, great effort is needed to educate Americans that cancer screening (checking for cancer in people with no symptoms) is key to early detection.

Another explanation for increased survival is improved treatment. Today, the traditional workhorses of cancer treatment—surgery, radiation, and chemotherapy—are being used in ways that are increasingly specific to the type of cancer involved. In fact, many cases of cancer now are being fully cured.

But is this the best we can do? What will the future bring? Hellman and Vokes, in their 1996 article in *Scientific American*, note that war often serves as a metaphor for cancer research. In 1971, two days before Christmas, President Richard M. Nixon signed the National Cancer Act, committing the United States to a "war" on cancer. Although the analogy is not perfect, Hellman and Vokes suggest that it can help us understand our current position with respect to cancer prevention, detection, and treatment. Looking at the "map" of cancer research after almost 30 years of "war," we can see that we have made some modest advances. But these successes do not reveal the tremendous developments that lie ahead of us by virtue of the new, strategic position we have achieved. In fact, most scientists expect that our newly gained understanding of the molecular basis of cancer will eventually give rise to a whole generation of exciting new techniques, not only for detecting and treating cancer but also for preventing it.

A key area of interest lies in learning how to exploit the molecular abnormalities of cancer cells to bring about their destruction. For example, understanding the role of oncogenes in the development of cancer suggests new targets for anticancer therapies. Some drug companies are working on drugs designed to shut down abnormal receptor proteins. Other potential targets are the aberrant proteins within the cytoplasm that transmit stimulatory signals even without being stimulated by surface receptors.

As in the case of oncogenes, a better understanding of the role of tumor suppressor genes in preventing runaway cell division may help scientists develop new therapies directed at these genes. For example, various studies have shown that introducing a normal tumor suppressor gene into a cell can help restore the cell to normalcy. Similarly, a therapy capable of restoring a cell's capacity for apoptosis would improve significantly the effectiveness of current cancer treatments. Even telomerase represents an important potential target for scientists looking for new and more powerful treatments for cancer. If telomerase could be blocked in cancer cells, their telomeres would continue to shorten with each division until their own proliferation pushed them into a genetic crisis and death.

One bold new research initiative that offers significant promise is the Cancer Genome Anatomy Project (CGAP). The project's goal is to identify all the genes responsible for the establishment and growth of human cancer. The work is based on a simple concept: Although almost every cell in the body contains the full set of human genes, only about one-tenth of them are expressed in any particular type of cell. Thus, different types of cells—for example, muscle cells and skin cells—can be distinguished by their patterns of gene expression.

Establishing for a particular cell the repertoire of genes expressed, together with the amount of normal or altered gene product produced by each expressed gene, yields a powerful "fingerprint" or "signature" for that cell type. Not unexpectedly, during the transformation of a normal cell to a cancer cell, this signature changes. Some changes are quantitative. That is, gene A may be expressed in both cells, but at greatly different levels, or it may be expressed in one cell but not the other. Other changes are qualitative: Gene B may be

expressed at the same level in both cells, but produce an altered product in the cancerous cell.

Scientists expect that being able to "read" these signatures—in other words, being able to compare the signatures of cells in their normal and cancerous states—will change cancer detection, diagnosis, and treatment in many exciting ways. Specifically, studying the exact sequence of molecular changes a cell undergoes during its transformation to a cancerous state will help scientists identify new molecular-level targets for prevention, detection, and treatment. One observation scientists have recently made is that cells surrounding an incipient tumor also may undergo changes that indicate that cancer is present. For example, early tobacco-induced molecular changes in the mouth may predict the risk of developing lung cancer, and cancers of the urinary tract may be signaled by molecularly-altered cells that are shed in the urine. Reading the signatures of these easily accessed cells may enable scientists to develop simple, non-invasive tests that will allow early detection of cancerous or precancerous cells hidden deep within the body.

Reading such signatures will also enhance the specificity of cancer diagnosis by allowing scientists to differentiate among tumors at the molecular level. By assessing the meaning of individual changes in a cell's signature, scientists will be able to determine which cancers are most likely to progress and which are not—a dilemma that confronts doctors in the treatment of prostate cancer—thereby allowing patients to avoid the harmful consequences of unnecessary treatment.

Finally, molecular fingerprinting will allow researchers to develop new treatments specifically targeted at cellular subtypes of different cancers. Often, patients suffering from tumors that by traditional criteria are indistinguishable, nevertheless experience quite different outcomes despite having received the same treatment. Research indicates that these different outcomes sometimes are related to the presence or absence of particular gene products. In the future, such molecular characteristics likely will be used to identify patients who would benefit from one type of treatment as compared with another.

The ultimate goal of such work, of course, is to push back the detection and diagnosis of cancer to its earliest stages of development. For the first time in the history of humankind, scientists can now envision the day when medical intervention for cancer will become focused at identifying incipient disease and preventing its progression to overt disease, rather than treating the cancer after it is well established.

Cancer and Society

But what does this mean for society? The financial costs of cancer loom large, not only for the individual but also for the community. The NCI estimates overall annual costs for cancer at about $107 billion. This cost includes $37 billion for direct medical costs, $11 billion for morbidity costs (cost of lost productivity), and $59 billion for mortality costs. Interestingly, treatment for breast, lung, and prostate cancers account for more than one-half of the direct medical costs.

Although early detection and successful treatment can reduce cancer deaths, the most desirable way to reduce them is prevention. In fact, scientists estimate that as many as one-half of the deaths from cancer in the United States and Europe, two areas with closely tracked cancer rates, could theoretically be prevented.

Nevertheless, the widespread persistence of unhealthful habits suggests that many Americans remain unconvinced about the power of prevention as a defense against cancer. Part of the reason may be that the only data we have about factors related to cancer are drawn from whole populations. These data cannot tell us who will develop cancer. Nor can they tell us whether healthful choices prevented its appearance in a particular individual.

Unhealthful habits also may persist because of the long time that elapses between the exposures that trigger the development of cancer and its actual appearance as disease. Conversely, there is a time lag between the institution of a beneficial personal habit (such as quitting smoking) or public policy (such as banning use of a known carcinogen) and its positive impact on personal and public health.

In their article "Strategies for Minimizing Cancer Risk," Willett, Colditz, and Mueller propose four levels on which to focus cancer prevention efforts. The first level is that of the individual. These authors argue that because most of the actions that can prevent cancer must be taken by individuals, dissemination of accurate information directly to the American public, together with peer support for behavioral changes, are critical.

A second level is health care providers, who are in a position to provide both counseling and screening to individuals under their care. Here, dissemination of accurate and timely information also is key.

A third level of prevention is the national level, where government agencies can impose regulations that help minimize the public's exposure to known carcinogens and implement policies that improve public health. Examples include regulating industries to cease using potent carcinogens and providing community facilities for safe physical activity.

Finally, a fourth level of prevention is at the international level, where the actions of developed countries can affect the incidence of cancer worldwide. Unfortunate examples of this include promoting the exportation of tobacco products and moving hazardous manufacturing processes to unregulated developing countries.

How do we think about devising and implementing measures to improve personal and public health in a pluralist society? One way to address this question is by attending to the ethical and public policy issues raised by our understanding and treatment of cancer.

Figure 9 A history of severe sunburns is strongly linked to the development of skin cancer later in life.

Ethics is the study of good and bad, right and wrong. It has to do with the actions and character of individuals, families, communities, institutions, and societies. During the last 2,500 years, Western philosophy has developed a variety of powerful methods and a reliable set of concepts and technical terms for studying and talking about the ethical life. Generally speaking, we apply the terms "right" and "good" to actions and qualities that foster the interests of individuals, families, communities, institutions, and society. Here, an "interest" refers to a participant's share in a situation. The terms "wrong" or "bad" apply to actions and qualities that impair interests. Often there are competing, well-reasoned answers to questions about what is right and wrong and good and bad about an individual's or group's conduct or actions.

Ethical considerations are complex, multifaceted, and raise many questions. In the United States, for example, we value protecting individuals from preventable harms. We support restrictions on who can purchase cigarettes and where smoking can occur. We inform pregnant women of the risks of drinking and smoking. However, we also value individual freedom and autonomy. We do not ban cigarettes outright; instead, we allow individuals over 18 years of age to take personal risks and be exposed to the related consequences. We permit pregnant women to buy and use liquor and cigarettes.

The inevitability of ethical tradeoffs is not simply a mark of the discussions in the United States. When considering differing health policy issues between and among countries, one cannot avoid encountering a pluralism of ethical considerations. Developing countries, whose health standards often differ from those in the United States, provide different cultural approaches to cancer and different standards for marketing and using tobacco and other known carcinogens. These different approaches raise a variety of ethical questions. For example, is there any legal and ethical way for people in the United States to prevent the widespread use of tobacco in other countries, a practice that contributes to the rise of lung cancer worldwide? Is there any legal and ethical way to govern

other choices of individuals (for example, poor diet and lack of exercise) that contribute to cancer?

Typically, answers to such questions all involve an appeal to values. A **value** is something that has significance or worth in a given situation. One of the exciting events to witness in any discussion in ethics in a pluralist society is the varying ways in which the individuals involved assign value to things, persons, and states of affairs. Examples of values that students may appeal to in discussions of ethical issues include autonomy, freedom, privacy, sanctity of life, protecting another from harm, promoting another's good, justice, fairness, relationships, scientific knowledge, and technological progress.

Acknowledging the complex, multifaceted nature of ethical discussions is not to suggest that "anything goes." Experts generally agree on the following features of ethics. First, ethics is a process of rational inquiry. It involves posing clearly formulated questions and seeking well-reasoned answers to those questions. Well-reasoned answers to ethical questions constitute **arguments**. Ethical analysis and argument, then, result from successful ethical inquiry.

Second, ethics requires a solid foundation of information and rigorous interpretation of that information. For example, one must have a solid understanding of cancer to discuss the ethics of requiring protective covering to be worn to prevent skin cancer. Ethics is not strictly a theoretical discipline but is concerned in vital ways with practical matters.

Third, because tradeoffs among interests are complex, constantly changing, and sometimes uncertain, there are often competing, well-reasoned answers to questions about what is right and wrong and good and bad. This is especially true in a pluralist society.

Public policy is a set of guidelines or rules that results from the actions or lack of actions of government entities. Government entities act by making laws. In the United States, laws can be made by each of the three branches of government: by legislatures (statutory law), by courts (case law), and by regulatory agencies (regulatory law). Regulatory laws are written by the executive branch of the government, under authorization by the legislative branch. All three types of law are pertinent to how we respond to cancer. When laws exist to regulate behavior, public policy is called **de jure** public policy.

Whether one makes public policy involves at least the following five considerations:

- the costs of implementing particular policies (including financial, social, and personal costs),
- the urgency of implementing a new policy,
- how effective a particular policy is likely to be,
- whether appropriate means exist to implement the policy, and
- social, cultural, and political factors.

For example, many argue that there is overwhelming evidence to support increased public policy restrictions on access to and use of cigarettes. Cigarette smoking is said to be linked to 85-90 percent of lung cancer cases. In 1998, 171,500 new cases of lung cancer were predicted. Of these, 160,100 were expected to end in death. Public policy prohibitions on cigarette use and access may be seen to satisfy four of the five criteria: (1) the cost of the policy would be minimal because cigarette access and use restrictions are in place, (2) the urgency of the situation is serious given the large number of deaths, (3) prohibiting purchase by

Figure 10 Where do we spend our money? A consequence of allowing unhealthful habits, such as smoking, is that public funds may be spent on cancer treatments instead of on other societal benefits, such as improved school facilities.

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minors and raising the prices (through taxation) are seen as effective, and (4) means are already in place for additional restrictions. The challenge in this era of high economic interest in cigarette production is the social, cultural, and political considerations (5).

It is important to recognize that sometimes the best public policy is *not* to enact a law in response to a controversy, but rather to allow individuals, families, communities, and societies to act in the manner they choose. Clearly, de jure public policy can only go so far in regulating people's behaviors. De jure public policy in the United States offers no match for the addictive power of nicotine and the marketing clout of the tobacco industry. In addition, any decline in cigarette use brought about by de jure public policy in the United States has been more than offset in recent years by a rapid increase of cigarette consumption elsewhere in the world.

When no laws exist to regulate behavior, public policy is called **de facto** (actual) public policy. With regard to lung cancer prevention programs, many think that other approaches are needed: improved general education and cultivation of an antismoking ethos. In any discussion of society's response to a social problem, it is important to think about other ways to address the problem.

Knowledge, choice, behavior, and human welfare. We can conclude that science plays an important role in assisting individuals to make choices about enhancing personal and public welfare. Science provides evidence that can be used to support ways of understanding and treating human disease, illness, deformity, and dysfunction. But the relationships between scientific information and human choices, and between choices and behaviors, are not linear. Human choice allows individuals to choose against sound knowledge, and choice does not necessarily lead to particular actions.

Nevertheless, it is increasingly difficult for most of us to deny the claims of science. We are continually presented with great amounts of relevant scientific and medical knowledge that is publicly accessible. We are fortunate to have available a large amount of convincing data about the development, nature, and treatment of particular cancers. As a consequence, we might be encouraged to think about the relationships among knowledge, choice, behavior, and human welfare in the following ways:

knowledge (what is and is not known) + choice = power

power + behavior = increased human welfare (that is, personal and public health)

One of the goals of this module is to encourage students to think in terms of these relationships, now and as they grow older.

Implementing the Module

The five activities in this module are designed to be taught either in sequence, as a supplement to your standard curriculum, or as individual activities that support or enhance your treatment of specific concepts in biology. The following pages offer general suggestions about using these materials in the classroom; you will find specific suggestions in the support material provided for each activity.

Goals for the Program *Cell Biology and Cancer* is designed to help students develop the following major goals associated with biological literacy: (1) to understand a set of basic scientific principles related to cancer as a cellular phenomenon, (2) to experience the process of inquiry and develop an enhanced understanding of the nature and methods of science, and (3) to recognize the role of science in society and the relationship between basic science and personal and public health.

Conceptual Organization of the Activities We have organized the activities to form a conceptual whole that moves students from an introduction to cancer (*The Faces of Cancer*), to an investigation of its biological basis (*Cancer and the Cell Cycle* and *Cancer as a Multistep Process*), to a discussion of how people evaluate claims about cancer (*Evaluating Claims About Cancer*), to a consideration of how understanding cancer can help people make decisions about issues related to personal and public health (*Acting on Information About Cancer*). Figure 11 illustrates the sequence of major concepts addressed by the five activities.

Figure 11 Conceptual Flow of the Activities

Activity	Major Concept
Activity 1 *The Faces of Cancer*	Cancer is a group of more than 100 diseases that develop across time. Cancer can develop in virtually any of the body's tissues, and both hereditary and environmental factors contribute to its development.
Activity 2 *Cancer and the Cell Cycle*	The growth and differentiation of cells in the body normally are precisely regulated; this regulation is fundamental to the orderly process of development that we observe across the life spans of multicellular organisms. Cancer develops due to the loss of growth control in cells. Loss of control occurs as a result of mutations in genes that are involved in cell cycle control.
Activity 3 *Cancer as a Multistep Process*	No single event is enough to turn a cell into a cancerous cell. Instead, it seems that the accumulation of damage to a number of genes ("multiple hits") across time leads to cancer.
Activity 4 *Evaluating Claims About Cancer*	Scientists use systematic and rigorous criteria to evaluate claims about factors associated with cancer. Consumers can evaluate such claims by applying criteria related to the source, certainty, and reasonableness of the supporting information.
Activity 5 *Acting on Information About Cancer*	We can use our understanding of the science of cancer to improve personal and public health. Translating our understanding of science into public policy can raise a variety of issues, such as the degree to which society should govern the health practices of individuals. Such issues often involve a tension between the values of preserving personal and public health and preserving individual freedom and autonomy.

Although we encourage you to use the activities in the sequence outlined in Figure 11, many of the activities can be taught individually, to replace or enhance a more traditional approach to the same or related content. Figure 12 provides recommendations for inserting the activities into a standard high school curriculum in biology.

Correlation to the *National Science Education Standards*

Cell Biology and Cancer supports teachers in their efforts to reform science education in the spirit of the National Research Council's 1996 *National Science Education Standards* (*NSES*). Figure 13 lists the specific content and teaching standards that this module primarily addresses.

Active, Collaborative, and Inquiry-Based Learning

The activities in this module are designed to offer students the opportunity to participate in active, collaborative, and inquiry-based learning in biology. But what do these terms mean? Despite their current popularity, many teachers think of active, collaborative, and inquiry-based learning rather generically. Defining these three key terms more specifically will provide a foundation on which we can build a detailed description of the instructional approach that the five activities in this module advocate and implement.

Conceptually the broadest of the three, **active learning** means that students are involved "in doing things and thinking about the things they are doing" (Bonwell and Eison, 1991, p. 2). These authors elaborate by listing the following characteristics typically associated with strategies that deserve to be labeled "active."

- Students are involved in more than listening.
- Instructors place less emphasis on transmitting information and more emphasis on developing students' skills.
- Students are involved in higher-order thinking (for example, analysis, synthesis, and evaluation).
- Students are engaged in activities (for example, reading, discussing, and writing).

Figure 12 Correlation Between Activities and Standard Curricula*

	Module Activity					Biology Textbook** Chapter									
Topic	1	2	3	4	5	DOL	AEE	LS	Blue	Green	Human	VL	P & E	Modern	TLS
biology of cancer	•	•	•			11	22	10	9	7	13 essay	6, 33	9	11	40
cell cycle and regulation of cell division		•	•			11	22	7	9	5	13 essay	6	6	8	5
mutation		•	•			13	10	8	13	8	12 essay	6,7, 8	9	10, 12	9
cancer and personal and public health				•	•	–	–	24	–	24	16	16	18	23	–

*The table indicates where topics addressed in the module are covered in a variety of current high school textbooks.

**DOL = *Biology: The Dynamics of Life* (Glencoe)
AEE = *Biology: An Everyday Experience* (Glencoe)
LS = *Biology: Living Systems* (Glencoe)
Blue = *BSCS Biology: A Molecular Approach* (D.C. Heath and Co./McDougal-Littel)
Green = *BSCS Biology: An Ecological Approach* (Kendall/Hunt)
Human = *BSCS Biology: A Human Approach* (Kendall/Hunt)
VL = *Biology: Visualizing Life* (Holt, Rinehart, Winston)
P & E = *Biology: Principles & Explorations* (Holt, Rinehart, Winston)
Modern = *Modern Biology* (Holt, Rinehart, Winston)
TLS = *Biology: The Living Science* (Prentice Hall)

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Figure 13 Correlation to the *National Science Education Standards*

The Content Standards	
Standard A: As a result of activities in grades 9–12, all students should develop abilities necessary to do scientific inquiry and understandings about scientific inquiry.	**Correlation to *Cell Biology and Cancer***
• Identify questions and concepts that guide scientific investigations.	Activities 2, 3, and 4
• Design and conduct scientific investigations.	Activity 4
• Use technology and mathematics to improve investigations and communications.	Activity 3
• Formulate and revise scientific explanations and models using logic and evidence.	Activities 2, 3, and 4
• Recognize and analyze alternative explanations and models.	Activity 3
• Communicate and defend a scientific argument.	Activity 4
• Understandings about scientific inquiry.	Activities 2, 3, and 4
Standard C: As a result of their activities in grades 9–12, all students	**Correlation to *Cell Biology and Cancer***
should develop understanding of the cell.	
• Cells store and use information to guide their functions.	Activities 2 and 3
• Cell functions are regulated.	Activity 2
should develop understanding of the molecular basis of heredity.	
• In all organisms, the instructions for specifying the characteristics of the organism are carried in the DNA.	Activities 2 and 3
• Changes in DNA occur spontaneously at low rates.	Activities 2 and 3
should develop understanding of the interdependence of organisms.	
• Human beings live within the world's ecosystems.	Activity 5
Standard E: As a result of activities in grades 9–12, all students should develop abilities of technological design and understandings about science and technology.	**Correlation to *Cell Biology and Cancer***
• Science often advances with the introduction of new technologies.	Activity 2
• Creativity, imagination, and a good knowledge base are all required in the work of science and engineering.	Activities 1–5
Standard F: As a result of activities in grades 9–12, all students should develop understanding of	**Correlation to *Cell Biology and Cancer***
• personal and community health.	Activities 1, 4, and 5
• natural and human-induced hazards.	Activities 1, 4, and 5

• science and technology in local, national, and global challenges.	Activity 5
Standard G: As a result of activities in grades 9–12, all students should develop understanding of	**Correlation to *Cell Biology and Cancer***
• science as a human endeavor.	Activities 2 and 4
• nature of scientific knowledge.	Activities 2, 3, and 4
• historical perspectives.	Activity 2
The Teaching Standards	
Standard A: Teachers of science plan an inquiry-based science program for their students. In doing this, teachers	**Correlation to *Cell Biology and Cancer***
• develop a framework of yearlong and short-term goals for students.	Each activity provides short-term objectives for students. Figures 11 (Conceptual Flow of the Activities) and 17 (Timeline for Teaching the Module) also help teachers plan.
• select science content and adapt and design curriculum to meet the interests, knowledge, understanding, abilities, and experiences of students.	Using the module helps teachers update their curriculum in response to their students' interest in this topic.
• select teaching and assessment strategies that support the development of student understanding and nurture a community of science learners.	The focus on active, collaborative, and inquiry-based learning in the activities helps teachers meet this standard.
Standard B: Teachers of science guide and facilitate learning. In doing this, teachers	**Correlation to *Cell Biology and Cancer***
• focus and support inquiries while interacting with students.	All of the activities in the module encourage and support student inquiry.
• orchestrate discourse among students about scientific ideas.	All of the activities in the module promote discourse among students.
• challenge students to accept and share responsibility for their own learning.	All of the activities in the module challenge students to accept and share responsibility for their learning.
• recognize and respond to student diversity and encourage all students to participate fully in science learning.	Combining the 5E instructional model with active, collaborative learning is an effective way of responding to the diversity of student backgrounds and learning styles.
• encourage and model the skills of scientific inquiry, as well as the curiosity, openness to new ideas and data, and skepticism that characterize science.	Annotations for the teacher that occur throughout the activities provide many suggestions for how teachers can model these attributes.
Standard C: Teachers of science engage in ongoing assessment of their teaching and of student learning. In doing this, teachers	**Correlation to *Cell Biology and Cancer***
• use multiple methods and systematically gather data about student understanding and ability.	Each activity has a variety of assessment components embedded within its structure. Annotations draw teachers' attention to these opportunities for assessment.

• analyze assessment data to guide teaching.	Annotations provide answers to questions that can help teachers analyze student feedback. The annotations also suggest ways for teachers to change their approach to students, based on that feedback.
Standard E: Teachers of science develop communities of science learners that reflect the intellectual rigor of scientific inquiry and the attitudes and social values conducive to science learning. In doing this, teachers	**Correlation to *Cell Biology and Cancer***
• display and demand respect for the diverse ideas, skills, and experiences of all students.	The answers provided in the annotations for teachers model these qualities.
• nurture collaboration among students.	All of the activities are designed to be completed by students working in collaborative teams.
• structure and facilitate ongoing formal and informal discussion based on a shared understanding of rules of scientific discourse.	All of the discussions in the activities model the rules of scientific discourse.
• model and emphasize the skills, attitudes, and values of scientific inquiry.	The annotations for teachers provide many suggestions about how to model these skills, attitudes, and values.

- Instructors encourage students' exploration of their own understandings, attitudes, and values.

Most teachers endorse the use of active learning. We know intuitively, if not experientially and explicitly, that learning does not occur through a process of passive absorption. But often we do not realize *how active* students must be for real learning to occur. Typically, the answer to this question is *more active* than we might expect.

The activities in this module were designed with the following assumptions about active learning (BSCS, 1999):

1. An activity promotes active learning to the degree to which *all students*, not simply a vocal few, are involved in mental processing related to the content.
2. An activity promotes active learning to the degree that it offers *extended opportunities* for students to become personally engaged with the content.
3. An activity promotes active learning to the degree that it involves students in thinking *deeply* about content.

The activities also make extensive use of **collaborative learning**. Most often occurring within the context of group work, collaborative and cooperative learning currently enjoy "favorite child" status among the many strategies available to teachers. Teachers are using group approaches across disciplines, for in- and out-of-class assignments, with large and small classes, and with beginning and advanced students. In fact, you will often find that collaborative activities go hand-in-hand with active learning.

Collaborative and cooperative learning, both with long theoretical and empirical histories, come out of different academic traditions, operate on different premises, and employ different strategies. But both approaches share a fundamental commitment to the notion that students learn from and with each other, "learning through joint intellectual effort," according to one expert (Brody, 1995, p. 134). In the interest of brevity, we will leave alone the finer distinctions between the two, offering in this curriculum a mix of strategies that put students together and engage them in tasks that encourage learning in collective contexts.

Finally, the activities in the module use **inquiry-based strategies**. All truly inquiry-based activities share the characteristics of active learning. In addition, inquiry-based strategies emphasize discovery:

the process of observation, followed by analysis, that leads to explanation, to conclusion, or to the next question. Note that an activity need not involve students in active experimentation to be fundamentally an inquiry experience.

More than active or collaborative learning, inquiry-based strategies attempt to teach students how biologists see the world, how they think about what they see, and how they draw conclusions that are consistent with observations and current knowledge. Such strategies say to the student, in effect, "*This* is science as a way of knowing."

The 5E Instructional Model

The activities in the module also have been designed using an instructional model to organize and sequence the experiences offered to students. This model, called the "5E model," is based on **constructivism**, a term that expresses a view of the student as an active agent who "constructs" meaning out of his or her interactions with events (Perkins, 1992). According to this view, rather than passively absorbing information, the student redefines, reorganizes, elaborates, and changes his or her initial understandings through interactions with phenomena, the environment, and other individuals. In short, the student interprets objects and phenomena and then internalizes this interpretation in terms of previous experiences.

A constructivist view of learning recognizes that the development of ideas and the acquisition of lasting understandings take time and experiences (Saunders, 1992). In the typical classroom, this means that fewer concepts and subjects can be covered during the school year or, in this case, in five days of instruction. Nevertheless, research suggests that students who are given time and opportunity to thoroughly grasp a small number of important concepts do better on traditional tests than students who are exposed briefly to a large number of ideas (Sizer, 1992; Knapp, 1995). In fact, the intensive thinking involved in constructing a thorough understanding of a few major ideas appears to benefit all students, regardless of ability.

Figure 14 illustrates the key components of the 5E model, so-called because it takes students through five phases of learning that are easily described using five words that begin with the letter "E": Engage, Explore, Explain, Elaborate, and Evaluate.

This instructional model allows students to share common experiences related to cancer, to use and build on prior knowledge, to construct meaning, and to assess continually their understanding of a major concept. It avoids excessive use of lecture because research shows that 10 minutes of lecture is near the upper limit of comfortable attention that students give to lecture material, whereas the attention span in an investigative activity is far longer (Project Kaleidoscope, 1991). In the 5E model, the teacher acts as facilitator and coach much more frequently than he or she acts as the disseminator of information.

The following paragraphs illustrate how the 5Es are implemented across the activities in this module. They also provide suggestions about effective teaching behaviors that help students experience each phase of the learning cycle.

Activity 1, *The Faces of Cancer*, serves as the Engage phase of instruction for the students. This phase of the model initiates the learning sequence and introduces the major topic to be studied. Its primary purpose is to capture the students' attention and interest. The activity is designed to make connections between past and present learning experiences and to anticipate upcoming activities. By completing it, students should become mentally engaged in the topic of cancer and should begin to think about how it relates to their previous experiences. Successful engagement results in students who are intrigued by the concepts they are about to study in depth.

The second and third activities in the module, *Cancer and the Cell Cycle* and *Cancer as a Multistep Process*, serve in a broad sense as the Explore and Explain phases of the model. Activity 2 begins with an exercise designed to provide students with a common experience to build on as they actively explore the cell cycle and growth control in normal and abnormal cells. Subsequent events in Activities 2 and 3 move students into the Explain phase of the model. During this phase, students develop an

Figure 14 The Key Components of the 5E Model

Phase	What the Teacher Does That Is	
	***Consistent* with the 5E Model**	***Inconsistent* with the 5E Model**
Engage	Creates interest Generates curiosity Raises questions Elicits responses that uncover what students know or think about the concept/subject	Explains concepts Provides definitions and answers States conclusions Provides premature answers to students' questions Lectures
Explore	Encourages students to work together without direct instruction from teacher Observes and listens to students as they interact Asks probing questions to redirect students' investigations when necessary Provides time for students to puzzle through problems Acts as a consultant for students	Provides answers Tells or explains how to work through the problem Tells students they are wrong Gives information or facts that solve the problem Leads students step-by-step to a solution
Explain	Encourages students to explain concepts and definitions in their own words Asks for justification (evidence) and clarification from students Formally provides definitions, explanations, and new labels Uses students' previous experiences as the basis for explaining concepts	Accepts explanations that have no justification Neglects to solicit students' explanations Introduces unrelated concepts or skills
Elaborate	Expects students to use formal labels, definitions, and explanations provided previously Encourages students to apply or extend concepts and skills in new situations Reminds students of alternative explanations Refers students to existing data and evidence and asks, "What do you already know?" "Why do you think . . . ?"	Provides definitive answers Tells students they are wrong Lectures Leads students step-by-step to a solution Explains how to work through the problem
Evaluate	Observes students as they apply new concepts and skills Assesses students' knowledge and/or skills Looks for evidence that students have changed their thinking or behaviors Allows students to assess their own learning and group-process skills Asks open-ended questions, such as "Why do you think . . . ?" "What evidence do you have?" "What do you know about x?" "How would you explain x?"	Tests vocabulary words, terms, and isolated facts Introduces new ideas or concepts Creates ambiguity Promotes open-ended discussion unrelated to concept or skill

explanation for the biological basis of cancer. Explain activities give students opportunities to articulate their developing conceptual understanding or to demonstrate particular skills or behaviors. This is where the teacher introduces terms such as "oncogenes" and "tumor suppressor genes." Keep in mind, however, that these activities are still *student*-centered. That is, the students are developing their own explanations for the development of cancer. Here, the teacher's role is to guide students so that they have ample opportunity to develop a more complete understanding of the biological basis of cancer. Students ultimately should be able to explain their understanding of cancer by bringing

together their experiences, prior knowledge, and vocabulary.

During the Elaborate phase of the model, exemplified in this module by Activity 4, *Evaluating Claims About Cancer*, students are challenged to extend their understanding of cancer. Through a new set of questions and experiences, students develop a deeper, broader understanding of the topic, obtain more information about areas of interest, and refine their scientific and critical-thinking skills. A teacher's primary goal in this phase of the model is to help students articulate generalizations and extensions of concepts and understandings that are relevant to their lives.

Finally, Activity 5, *Acting on Information About Cancer*, serves as the Evaluate activity for the program. At this point, it is important that students see they can extend and apply their understanding of cancer to the real world. It also is important that they receive feedback on the adequacy of their explanations and understandings. Evaluate activities are complex and challenging, and Activity 5 will stretch your students' abilities to listen, think, and speak.

Using the *Cell Biology and Cancer* CD-ROM in the Classroom

The *Cell Biology and Cancer* CD-ROM is a tool, like an overhead projector or a textbook, that you can use to help organize your use of the module, engage student interest in learning, and help orchestrate and individualize instruction. The CD-ROM contains the following major resources:

- introductions to the National Institutes of Health and the National Cancer Institute;
- printable files of this module;
- printable files of the print-based alternatives for Activities 2, 3, and 5;
- the video clips and animations required to teach Activity 2, *Cancer and the Cell Cycle*;
- the simulation required to teach Activity 3, *Cancer as a Multistep Process*; and
- the video clips and reference database required to complete Activity 5, *Acting on Information About Cancer*.

The CD-ROM runs on Apple Macintosh and IBM-compatible personal computers. The recommended requirements for a Macintosh computer are the following: OS 7.1 operating system or higher, 68030 or Power Mac processor, 256 color monitor or higher, 8 megabytes RAM, QuickTime 4 for Macintosh, and a 2x CD-ROM.

The recommended requirements for IBM-compatible computers are the following: Windows 95 operating system or higher, Pentium 60 processor or higher, 256 color monitor or higher, 8 megabytes RAM, Soundblaster or Windows Sound System-compatible card, QuickTime 4 for Windows, and a 2x CD-ROM.

To use the CD-ROM, load it into the CD-ROM drive as you would any other CD. If you do not have QuickTime 4 loaded on your computer, you will see a dialogue box that will ask if you want to install it. Click Yes to automatically load the program. Then, follow the installation instructions shown in Figure 15.

Figure 15 Loading Instructions for the *Cell Biology and Cancer* CD-ROM

IBM-Compatible Computers

Place the CD in the CD-ROM drive and close the door. The CD should automatically launch the program.

If you have turned off the autorun feature on your CD-ROM drive, you must run the setup program the first time you use the software. Click Start | Run and type the following into the dialog box:

d:\setup.exe (change "d:\" depending on the letter of your CD-ROM drive)

If you want to run the software without ejecting and re-inserting the disk each time you use the program, do one of the following:

- Click Start | Programs | NIH Supplements | NIH CD-ROM
- Click Start | Run and type the following in the dialog box:
  d:\hsplayer\hsplayer.exe home.stk
  (change "d:\" if necessary). Click OK.

Macintosh Computers

Place the CD in the CD-ROM drive and close the door.

Open the CD-ROM, then click on the NCI icon.

Network Installation

A network installation of the entire program requires up to 250 to 450 megabytes of disk space. Performance of the videos will depend on the network speed and the processor speed of client stations. Each client computer must have QuickTime 4 or higher installed.

1. Place the disk in the CD-ROM drive and click on Quit if the program opens automatically.
2. Create a folder on the network or local drive where you want to install the application and name it Cancer.
3. Copy all the folders and files in the root directory of the CD-ROM into the new folder. Note: Macintosh users cannot see files from the PC format on the CD-ROM and vice versa. If you run both platforms from your network, you will need to copy files from the CD to the network twice, once from a network PC and once from a network Mac. If you have room, create two complete copies of the software in different folders, one for each platform. Because users will see both Mac and PC files on the network, be sure that Mac users open only the Mac files and PC users open only the PC files.
4. To run the application, follow the procedures described here for IBM-compatible or Macintosh computers by locating the local or network copy of the desired HyperStudio player files.

The ideal use of the CD-ROM requires one computer for each student team; the installation instructions explain how to make the information available over a network. However, if you have only one computer and CD-ROM drive available, you can still use the CD (for example, by using a suitable display device to show animations or videos to the whole class or by rotating teams through a computer station to access CD-ROM-based resources).

If you do not have the facilities for using the CD-ROM in your classroom, a print-based alternative for each activity that requires the CD is available for printing from the CD-ROM. To use this version, you will need to print out the activity lesson plan and its associated masters.

Before you use this CD-ROM or any other piece of instructional software in your classroom, it may be valuable to identify some of the benefits you expect the software to provide. For example, Roblyer (1997) suggests four major ways that instructional multimedia software can benefit students and teachers. First, well-designed multimedia software can **help motivate students**, help them enjoy learning, and help them want to learn more. Multimedia programs offer users a rich, interesting, and compelling environment in which to explore and learn, and it rewards users with a broader and more complex set of sensory experiences than print-based resources can provide. Well-designed multimedia resources can enliven content that students otherwise may perceive as dull and uninteresting. The video clips and animations provided on the *Cell Biology and Cancer* CD offer students this benefit. Because multimedia programs often provide nonlinear access to a rich array of information and stimulation, they also can encourage reluctant students to immerse themselves in a topic, creating, in effect, a positive feedback loop in which students learn as they "go their own way," wherever their interest or curiosity takes them.

Second, well-designed multimedia software also **offers unique instructional capabilities**. For example, such software can stimulate students to explore topics in greater depth and in more different dimensions than students often are willing or able to pursue. The simulation provided for Activity 3 and the reference database that supports Activity 5 have this effect. This benefit is related to the first,

but it deepens and intensifies learning rather than stimulates students to investigate content they otherwise would not investigate. Part of this benefit derives from the power such software has to provide essentially immediate access to a wealth of ever more detailed and complex information on a topic, all presented in interesting and unusual ways. Part of the benefit, however, derives from the software's very design: A well-designed user interface provides an easy-to-use navigation system, stimulates curiosity, and encourages exploration of related areas.

Completing activities using instructional software can help students learn to organize and be responsible for their own learning rather than depend entirely on the teacher for direction and support. This goal is commonly cited by teachers and employers, most of whom explicitly desire students and employees who are self-directed and can structure and execute work independently.

Multimedia software can offer students learning experiences that are closer to actual field experiences than the experiences print-based resources offer. The videos that support Activity 5 allow students to listen to people advocating real positions on the topic under investigation. Although the student's experience of the situation in Activity 5 is vicarious, it is more realistic and memorable than the comparatively static and unchanging experience that a textbook treatment of this topic would offer. Because it engages more senses than simply sight, and because it requires more skills than simply understanding what one reads, well-designed instructional software also addresses many different learning styles and serves the needs of a wider population of students than most print-based resources.

Third, multimedia software can provide teachers with **support for experimenting with new instructional approaches**. The educational system in the United States is struggling to improve its ability to prepare students for the complex, collaborative, technology-rich workplace they will enter when they leave school. Technology can make possible new approaches to teaching in the classroom. For example, by moving the responsibility for organizing learning from the teacher to the student, instructional software can help teachers move into the role of observer and facilitator of learning rather than dispenser of information. As students work independently or in small teams, teachers can circulate throughout the room, listening to students interact with one another, asking and answering questions, and challenging students to consider alternative lines of research and analysis. These behaviors are very different from the typical ones teachers are engaged in when they carry the primary responsibility for delivering and explaining content.

Instructional software also can be an effective tool for helping teachers organize discussions of controversial issues in the classroom. In Activity 5 in this module, using videos to present conflicting positions lends greater credibility to these positions than they may have if they were presented by the teacher. It also depersonalizes the positions, allowing both teachers and students to focus on the substance of the issues rather than on the controversy itself.

Software programs on CD-ROM also offer teachers the opportunity to expand and enrich the number and depth of research-based projects they assign students, and to increase the scope and difficulty of problem- or case-based activities they use in their classrooms. Although basic mathematic and communication skills still are considered essential for students to develop, educators are becoming increasingly aware that curricula must place less emphasis on learning specific factual information and place more on the ability to locate and use information to solve problems and to think critically about issues. The reference database provided in support of Activity 5 allows teachers to involve students in problem-solving and locating and using information while teaching the basic skills students are expected to acquire.

Finally, well-designed instructional software can **increase teacher productivity**. There are a variety of ways such software can accomplish these goals, such as helping teachers with assessment, record keeping, and classroom planning and management. Instructional software such as the CD-ROM

enclosed with this module offers teachers the convenience of a full week of instruction that is stored and transported in the space of a single CD and this teacher's guide. Instructional software also can give teachers increased credibility in their students' eyes. Many of today's students have been raised in a technology-rich environment and often respond positively to the use of technology-based methods that streamline and enhance communication between teachers and students and, in so doing, increase the efficiency of both.

Organizing Collaborative Groups

All of the activities in this module are designed to be completed by groups of students working together. Although many of the specific steps can be completed by individual students working alone, this strategy will not stimulate the types of student-student interactions that are one of the goals of active, collaborative, inquiry-based learning. Therefore, we recommend that you organize collaborative groups of between two and six students each, depending on the number of computers equipped with CD-ROM drives you have available. Students in groups larger than this likely will have difficulty organizing the student-computer interactions equitably, which can lead to one or two students assuming the primary responsibility for the computer-based work. Although this type of arrangement can be efficient, it means that some students do not get the opportunity to experience the in-depth discovery and analysis that the enclosed CD-ROM was designed to stimulate.

If you are teaching all five activities as a unit, we recommend that you keep your students in the same collaborative groups for all of the activities. This will allow each group to develop a shared experience with the software and with the ideas and issues that the activities present. A shared experience also will enhance your students' perceptions of the activities as a conceptual whole. This will be particularly important in the activities toward the end of the module, as students consider some of the ethical and public policy implications of basic research related to cancer.

If your student-to-computer ratio is greater than six students to one computer, you will need to change the way you teach the module from the instructions in the activities. For example, if you have only one computer available, you may want students to complete the CD-based work across an extended time period. You can do this in several ways. The most practical way is to use your computer as a center along with several other centers at which students complete other activities. In this strategy, students would rotate through the computer center, eventually completing the CD-based work that you have assigned.

A second way to structure the activities if you only have one computer available is to use an overhead projection system to display the computer monitor onto a screen for the whole class to see simultaneously. Giving selected students in the class the opportunity to manipulate the program in response to class suggestions and requests can give students some of the same type of autonomy over their learning that they would gain if they were working with the CD in small teams. Activity 5 requires students to use the CD for extensive research; in this case, give the students printouts of the reference database to work from. This strategy, however, will not give the students an opportunity to interact personally with the CD. We recommend that you use this strategy only if you have no other options.

Dealing with Values and Controversial Topics

Instructors sometimes feel that the discussion of values is inappropriate in the science classroom or that it "detracts" from the learning of "real" science. The activities in this module, however, are based upon the conviction that there is much to be gained by involving students in analyzing issues of science, technology, and society. Society expects all citizens to participate in the democratic process, and our educational system must provide opportunities for students to learn to deal with contentious issues with civility, objectivity, and fairness. Likewise, students need to learn that science intersects with life in many ways. Opportunities to encounter and consider carefully some of these ways will reinforce and enrich those scientific principles that we desire to teach.

The activities on the CD provide a variety of opportunities for students to discuss, interpret,

and evaluate basic science and public health issues in the light of values and ethics. Many issues that students will encounter—especially those having to do with public policies that force people to protect themselves from agents known to cause cancer—are potentially controversial. How much controversy develops will depend on many factors, such as how similar your students are with respect to socioeconomic status, perspectives, value systems, and religious preferences. It also will depend on how you handle your role as facilitator of the discussion. Your language and attitude may be the most important factors to the flow of ideas and the quality of exchange among the students.

Neutrality may be the single most important characteristic of a successful discussion facilitator. The following suggestions may help you think about how to guide your students in discussions that balance factual information with feelings.

- Encourage your students to discover as much information about the issue as possible. Ask questions that will help your students distinguish between those components of an idea or issue that scientific research can answer and those components that are a matter of values. Students should understand the importance of accurate information to any discussion and should recognize the importance of distinguishing factual information from opinions.
- Keep the discussion relevant and moving forward by questioning or posing appropriate problems or hypothetical situations. Invite your students to respond to or build on each other's ideas. Avoid asking questions that have exact answers unless the facts are important to the integrity of the discussion. Encourage everyone to contribute, but do not force reluctant students into the discussion.
- Emphasize that everyone must be open to hearing and considering diverse views. Point out that we cannot make intelligent decisions if we close ourselves off from some viewpoints. Even if we cannot agree with or are offended by a viewpoint, we still must hear it so that we know that it exists and can consider it as we shape our own views.
- Use unbiased questioning to help the students critically examine all views presented. Help your students consider different points of view thoroughly by asking them to define the relevant arguments and counterarguments. Let the students help you promote the expression of alternative points of view.
- Allow for the discussion of all feelings and opinions. Avoid becoming a censor of views that are radical or shocking (as long as these views are consistent with the facts). When a student seems to be saying something for its shock value, see whether other students recognize the inappropriate comment and invite them to respond.
- Avoid seeking consensus on all issues. The multifaceted issues that the students discuss result in the presentation of divergent views, and students should learn that this is acceptable. In some cases, however, helping the group reach consensus on a compromise solution to a problem may demonstrate compromise as a powerful determinant of cooperative community action.
- Keep your own views out of the discussion. Experts in science education recommend that teachers withhold their personal opinions from students. The position of teacher carries with it an authority that might influence students. The danger also exists that the discussion might slip into indoctrination into a particular value position, rather than an exploration of divergent positions. Either result misses the point of the discussion. If your students ask what you think, you may wish to respond with a statement such as "My personal opinion is not important here. We want to consider your views."
- Acknowledge all contributions in the same even-handed manner. If the class senses that you favor one idea over another, you will inhibit open debate and discussion. For example, avoid praising the substance or content of comments. Instead, acknowledge the willingness of students to contribute by making such comments as "Thanks for that idea" or "Thanks for those comments." As you display an open attitude, a similarly accepting climate will begin to develop within the class.
- Create a sense of freedom in the classroom.

Remind students, however, that freedom implies the responsibility to exercise that freedom in ways that generate positive results for all. If necessary, remind them that there is a fine line between freedom and license. In general, freedom is a positive influence, whereas license usually generates negative results.

- Insist upon a nonhostile environment in the classroom. Do not allow your students to make *ad hominem* arguments (arguments that attack the person instead of the idea). Help your students learn to respond to ideas instead of to the individuals presenting those ideas.
- Respect silence. Reflective discussions often are slow. If you break the silence, your students may allow you to dominate the discussion.
- Finally, at the end of the discussion, ask your students to summarize the points that they and their classmates have made. Let your students know that your respect for them does not depend on their opinion about any controversial issue. If students feel that they must respond in particular ways to gain your approval, your class will not discuss issues openly and with forthrightness.

Following these general suggestions should help you stimulate meaningful student-to-student interaction with as little direct involvement by you as possible. Initially, some students may have difficulty responding without specific direction. It is important, however, that you resist the temptation to intervene extensively in the initial, sometimes uncomfortable phase of long silences and faltering responses. Unless students are given opportunities to evaluate ideas and values in the context of a larger problem, they may never learn to do so.

Assessing Student Progress

Because we expect this module to be used in a variety of ways and at a variety of points in an individual teacher's curriculum, we believe the most appropriate mechanism for assessing student learning is one that occurs informally at various points within the activities, rather than something that happens more formally just once at the end of the module. Accordingly, we have integrated a variety of specific assessment components throughout the activities within the module. These "embedded assessment" opportunities include one or more of the following strategies:

- performance-based activities (for example, developing media items or participating in a structured discussion of a potentially controversial issue);
- oral presentations to the class (for example, presenting experimental results); and
- written assignments (for example, answering discussion questions or writing short reports).

These strategies allow you to assess a variety of aspects of the learning process, such as students' prior knowledge and current understanding, problem-solving and critical-thinking skills, level of understanding of new information, communication skills, and ability to synthesize ideas and apply understanding to a new situation.

An assessment icon and an annotation that describes the aspect of learning you can assess using a particular strategy appear in the margin beside the step in which each embedded assessment occurs.

Student Activities

The heart of this module is the set of five activities that follow. These activities are the vehicles that we hope will carry important concepts related to cancer and personal and public health to your students. To review the concepts in detail, refer to Figure 11 in *Implementing the Module*.

As you scan the activities, you will find that each contains several major features.

At a Glance gives you a convenient overview of the activity.

- The **Focus** provides a one- to two-sentence summary of what students do.
- **Major Concepts** state the central idea(s) the activity is designed to convey.

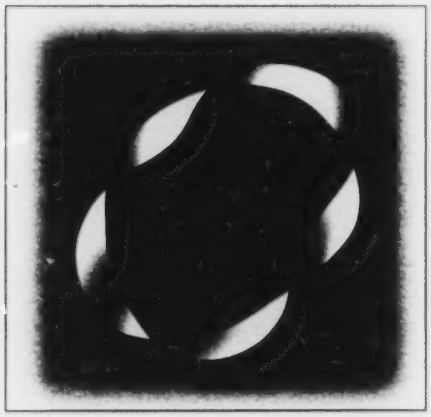

Figure 16 A Möbius strip is a one-sided, one-edged loop. Test this by making a loop with five twists. With a marker, draw a continuous line around the strip, starting at the seam. Your line should pass along "both" sides of the paper before you return to your starting point, even though you do not lift your marker off the paper as you draw. Then run your marker along the edge, again starting at the seam. You should see that the strip also contains only one edge. Loops with odd numbers of twists are Möbius strips; loops with even numbers of twists are not. In this module, we use a five-twist Möbius strip as a metaphor for the relationship between basic science and personal and public health.

- **Objectives** lists three to five specific understandings or abilities students should have after completing the activity.
- **Prerequisite Knowledge** alerts you to the understandings and skills students should have before beginning the activity.
- The **Basic Science-Public Health Connection** describes how the activity illustrates the relationship between basic science and personal and public health. The mission of the NIH is to "uncover new knowledge that will lead to better health for everyone." This mission statement recognizes that basic science and personal and public health are not separate issues; they are not even two sides of one issue (Figure 16). Rather, they are inextricably linked and form a powerful whole: Research into the basic processes of life leads inevitably to strategies for improving health, and questions about health trigger research into basic processes.

The **Introduction** places the activity in a context and provides a short overview of its key components.

Materials and Preparation provides instructions for collecting and preparing the materials required to complete the activity.

Procedure outlines the activity's steps and provides implementation suggestions and answers to questions. Annotations in the margins, identified by icons, provide specific hints about helping students see connections between basic science and personal and public health (the Möbius strip icon), assessing student understanding (the checkmark icon), and focusing students' attention on the activity's major concepts during its closing steps (the "completing-the-puzzle" icon).

Potential Extensions describes ways you can extend or enrich the activity.

All of the **Masters** required to teach the activities are located in a separate section at the end of the module.

Three of the activities (*Cancer and the Cell Cycle*, *Cancer as a Multistep Process*, and *Acting on Information About Cancer*) use the enclosed *Cell Biology and Cancer* CD-ROM. For information about using the CD, see the section "Using the Cell Biology and Cancer CD-ROM in the Classroom" in *Implementing the Module*. If you do not have enough computers equipped with CD-ROM drives available to conduct these activities with your students, you can use the print-based alternatives. To view and print the instructions and masters for these alternate activities, load the CD onto a computer and click the Print button on the main menu screen. The computer will display a screen showing the resources available for printing from the CD; click on the button labeled Non-CD Lesson Plan from the choices available for the relevant activity. This will reveal the lesson plan and the masters for the alternate, non-CD-based lesson. Click Print again to print these resources.

One activity (*Evaluating Claims About Cancer*) involves students in designing and executing their own experiments. Ordering and preparation instructions for the materials required are provided on page 70 and instructions for students appear on Master 4.5.

Figure 17 outlines a plan for preparing for and completing the five activities that follow. The page references in the caption indicate the pages on which you will find specific preparation instructions. The plan assumes you will teach the activities on consecutive days. If this is not your plan, adjust the timing of your preparation steps accordingly.

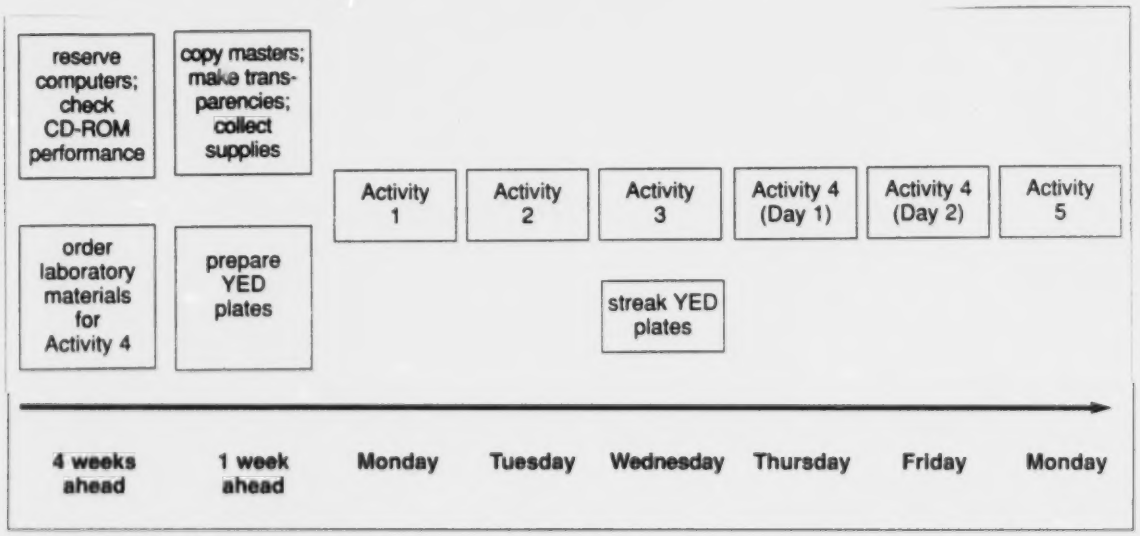

Figure 17 Timeline for teaching the module. Before you begin teaching this module, review this timeline. Instructions for computer set-up are on pages 28–29; instructions for laboratory preparation are on page 70; and instructions for preparing other required materials appear under Materials and Preparation in each activity.

Activity 1

The Faces of Cancer

At a Glance

Focus: Students participate in a role play about people who develop cancer, assemble data about the people's experiences with cancer, then discuss the generalizations that can be drawn from these data.

Major Concepts: Cancer is a group of more than 100 diseases that develop across time. Cancer can develop in virtually any of the body's tissues, and both hereditary and environmental factors contribute to its development.

Objectives: After completing this activity, students will

- understand that there are many types of cancer,
- recognize that the incidence of cancer increases with age,
- understand that some people inherit predispositions to particular types of cancer,
- understand that some people make choices that increase their risk for cancer; and
- be able to explain that a person's chance of surviving cancer increases with early detection and treatment.

Prerequisite Knowledge: None

Basic Science-Public Health Connection: This opening activity introduces cancer as a public health issue that can be systematically studied using the methods of science (for example, gathering and analyzing data).

Introduction

As described in *Understanding Cancer*, cancer is a group of diseases that are characterized by uncontrolled cell division. This uncontrolled division can compromise the function of an organism and ultimately may cause its death.

Each of us has a chance of developing cancer sometime in our life. On average, in the United States, men have a 1 in 2 lifetime risk of developing cancer and women a 1 in 3 risk. Many Americans, however, have a higher than average chance of developing particular forms of cancer. For example, smokers have a 10-fold higher risk of developing lung cancer compared with nonsmokers. Likewise, women who have a mother, sister, or daughter who has had breast cancer have about a 2-fold higher chance of developing breast cancer compared with women who do not have such a family history.

The National Cancer Institute estimates that approximately 8 million Americans alive today have a history of cancer. In 1998, more than 1 million new cases were diagnosed. In fact, cancer is the second leading cause of death in the United States, exceeded only by heart disease.

This activity introduces the module by reminding students that cancer is a public health issue in the United States. In the activity, students participate in a role play based on the relative incidence of various types of cancer in the U.S. population for a recent year. As part of the role play, each student receives information about one person who develops cancer. Students work in groups, discussing the relevant events of each person's life; as a class, they assemble data about the population as a whole. Students examine the data the class assembles and draw a set of important generalizations about cancer from them. These generalizations set the stage for subsequent activities in the module in which students learn how cancer develops and consider how claims about factors alleged to cause cancer are evaluated and acted upon.

Materials and Preparation

You will need to prepare the following materials before conducting this activity:

- identity envelopes (make 1 envelope per student)

  To make a set of identity envelopes for your class, first make one complete copy of Master 1.1, *The Faces of Cancer*. Cut each page along the lines to create five separate pieces of paper. Paste the piece that names and describes each fictitious person onto the front of an envelope, then place the remaining four pieces (labeled "0–19 years," "20–39 years," "40–59 years," and "60+ years") inside the envelope, in order from the earliest period of life to the latest.

- Master 1.2, *Team Summary* (make 1 copy per student)
- Master 1.3, *Drawing Conclusions from the Faces of Cancer* (make 1 copy per student)
- Master 1.4, *Summary Profile of the Faces of Cancer* (make 1 overhead transparency)

Procedure

With 1 in 3 Americans developing cancer in their lifetimes, it would not be unusual if one or more of your students is personally involved with cancer. It may be that the child's parent, family member, or even the child has or has had cancer. For some of these students, the topic of cancer may be disturbing. Because of this, we suggest that you watch your students for signs of discomfort with the topic (for example, reluctance to begin the activity, unusual quietness or reticence) and provide appropriate support.

It may be useful to begin the activity by asking students to indicate with a show of hands whether they have had an experience with cancer and offer those who raise their hands the opportunity to share it with the class. Emphasize that students also may keep this information private, if they so choose. During the team work, you may want to approach those students who raised their hands and assure yourself that they are handling the activity well.

If you have a student who is having serious difficulty with the topic, you may want to offer him or her a learning alternative to completing the activities in the module.

1. **Introduce the activity by asking students to count off in sets of 6 (that is, 1, 2, 3, 4, 5, 6, and so on) and to write down their numbers.**

Counting off in this manner sets up the demonstration of statistics that occurs in Step 2 and also identifies the teams into which students will organize in Step 5.

2. **Explain that the American public often is presented with statistics about various characteristics of the U.S. and world populations. Sometimes a good way to get a sense of what such statistics mean is to express them in terms of a group of real people. To illustrate this, conduct the following exercise.**

   - **Ask all the students who are number 2s, 3s, 5s, or 6s to stand. Explain that if the population in this class is representative of the American population, approximately 6 in 10 (or in this case, 4 in 6) of the people in the room will have children.**

     The proportion of U.S. citizens who have children is a rough estimate based on data from 1994 indicating that 42 percent of women aged 15 to 44 do **not** have children.

   - **Invite the students who are standing to sit, then ask all the students who are number 3s or 6s to stand. Explain that if the class population is representative, approximately 3 in 10 (or in this case, 2 in 6) of the people in the room will be involved in an alcohol-related automobile accident sometime in their lifetimes.**

     A fact sheet published by the National Highway Traffic Safety Administration in 1997 (Traffic Safety Facts 1997, National Highway Traffic Safety Administration, http://www.nhtsa.dot.gov/people/ncsa/ovrfacts.htm) estimated that about 3 in every 10 Americans would be involved in an alcohol-related motor vehicle accident at some time in their lives.

   - **Invite the students who are standing to sit, then ask all the students who are number 1s or number 4s to stand. Explain again that if the class population is representative, approximately 1 in 3 (or in this case, 2 in 6) of the people in the room will develop cancer sometime during their lifetimes. Ask students if this statistic surprises them.**

     Answers will vary.

   - **Finally, ask about one fourth of the students who are standing to sit (for example, if 10 students are standing, ask 2 or 3 to sit). Explain that the number of students left standing represents the approximate percentage of the U.S. population who will die of cancer (about 25 percent). Note that the work of scientists and health care professionals across many years has increased the gap between the number of people who develop cancer and the number of people who die from it, and ask your students what factors they think are contributing to this increased gap.**

In March 1998, the National Cancer Institute, American Cancer Society, and Centers for Disease Control and Prevention announced that cancer incidence and death rates for all cancers combined and for most of the top 10 sites declined between 1990 and 1995, reversing an almost 20-year trend of increasing cancer cases and death rates in the United States. Point out that this provides evidence that cancer research has paid off in thousands of human lives saved. Suggest that some students may want to consider a career in cancer research.

Students likely will answer that it is the result of increased prevention, earlier detection, and improved treatment.

3. **Invite the students who are still standing to sit, then ask the class whether there is any way to know who will develop cancer and when.**

   Students may answer that there is no way to know for sure, but, in general, old people, people who smoke, and people exposed to excessive radiation develop cancer. Accept all reasonable answers without comment; the purpose of this questioning is to encourage students to express what they already know about cancer and to highlight the fact that there is no definitive way to know who will develop cancer. If students make questionable claims about risk factors or other aspects of cancer, you may wish to respond that many claims are made about cancer and then ask students how they could investigate such claims. You may also wish to point out that Activity 4, *Evaluating Claims About Cancer*, addresses this question.

4. **Explain that in this activity, students will learn more about who develops cancer, when, and why, by assuming the identities of 30 [insert the number of students in your class] fictitious people who develop cancer and building a profile of some of the key events in these people's lives.**

   Current statistics reveal that only 1 in 3 Americans will develop cancer during their lifetimes. In this activity, however, each of the 30 fictitious people develops cancer. The activity is structured in this way to offer all students similar experiences and to provide maximum richness and variety to the stories of cancer the students encounter. Students will be reminded of the 1 in 3 risk of developing cancer when they complete the questions on the bottom of *Drawing Conclusions* (see Step 18).

5. **Direct the students to organize into teams based on the number they received during the count-off (all students with number 1 should form one team and so on).**

   Students will work in teams of four to five throughout the activities in the module. To ensure that students working together as members of one team have a common foundation of experience and understanding, we recommend that you keep students in the same teams for all of the activities.

6. **Distribute one identity envelope to each student and explain that the outside of the envelope contains a description of the person that student is to become. Ask students to read the descriptions on the envelopes they receive and share who they are with the other members of their teams. Ask students *not* to open their envelopes at this time.**

   We suggest that you do not try to match male students with male names and female students with female names. Instead, distribute the envelopes randomly throughout the class. This strategy simplifies the

process of distributing the envelopes and avoids the problem that your class may contain a different number of males and females than the identity envelopes do.

To make the activity fun, encourage students to read the description of the person they have "become" to themselves, then introduce themselves (in first person) to the other members of their team. As students move through the activity, encourage them to "tell" their stories to the rest of the team, using first person language and representing the person they have become as realistically as they can.

Tip from the field test. Before distributing the envelopes, you may wish to explain that some students will be asked to assume the identities of people quite different from themselves (for example, a different sex, or ethnic or cultural group). Explain that this is an inevitable consequence of the activity's structure and ask all students to do the best job they can representing the people whose identities they have assumed.

7. **While students are discussing their new identities, distribute one copy of Master 1.2, *Team Summary*, to each student.**

8. **Explain that the students' task in the next few minutes will be to use the *Team Summary* to summarize information about the lives of the fictitious people in their team. Point out that the descriptions they just read contained information about whether each person had a history of cancer in his or her family. Ask students to use this information to complete Section 1, Family History, on their *Team Summary*.**

   Give the students 1–2 minutes to complete this task. If necessary, explain that having a "history of cancer in the family" means having a biological relative (grandparent, parent, sibling, aunt, or uncle) who has or has had cancer.

9. **Explain that inside each envelope is a set of four cards that provide additional information about each person's life. Direct students to remove the cards from their envelopes and place them *face down* on the desks in front of them so that the cards are in sequence, with the card labeled "0–19" years on top and the card labeled "60+ years" on the bottom.**

   Each student should have four cards. Some of the fictitious people were "born" in the early 1900s and are "old" enough to be 70 or 80; others were born much later (for example, in the 1970s or 1980s). Nevertheless, we have extended these peoples' lives to 60+ years, even though this time stretches well into the 21st century. This approach allows the activity to illustrate a wide range in choices and health care options across the 20th century. The approach also gives each student a chance to have four cards and participate to the end of the activity.

10. **Invite the students to turn over and read the cards labeled "0–19." Give the students a few minutes to share the information they learn with the**

other members of their teams, then challenge them to use this information to complete the "0–19 years" column in Section 2, Cancer History, of their *Team Summary*.

To heighten the activity's drama, do not allow students to read all of their cards at once. Insist that the students in each team progress through the life stages in sequence together.

As students begin to read their cards, they may need help understanding how to fill in Section 2 of the *Team Summary*.

11. **Instruct students to turn over the rest of their cards in sequence, share the information the cards contain, then use this information to complete Section 2 of their *Team Summary*. Challenge the students to look for patterns or trends in the data they are collecting and explain that when the class pools all of its data, the students will be able to determine the degree to which the patterns they see in their team's data also appear in the pooled data.**

    The black dot that appears on one of the four cards for each person shows when mutations may have occurred that eventually contributed to the development of cancer. Some students may ask what this dot represents. **Do not explain the dot at this point.** Respond that students will discover the dots' significance at the end of the activity (see Step 16).

12. **After the students complete Section 2 of their *Team Summary*, ask them if they noticed any choices or other risk factors that may be related to the cancer people developed. Instruct students to go back through their cards to identify these factors, then list them in Section 3, Possible Risk Factors, of their *Team Summary*.**

    Some of these risk factors are smoking, sun exposure, high fat diet, early sexual activity, and genetic predisposition for cancer. A major factor that is not specifically noted is aging. The explanation for increased incidence of cancer with aging is explored in Activity 3, *Cancer as a Multistep Process*.

13. **As the teams complete their summaries, distribute one copy of Master 1.3, *Drawing Conclusions from the Faces of Cancer*, to each student.**

14. **Display the transparency that you prepared from Master 1.4, *Summary Profile of the Faces of Cancer*, and explain that you will complete the table as the teams share the information they have collected. Explain that as you complete each row of the table, you will give the teams 2–3 minutes to discuss and record a conclusion they can make from the pooled data.**

    To illustrate, ask each team to report how many people in that team did and did not have a history of cancer in their families. Then, ask the students what pattern they see in the pooled data and what conclusion

it leads them to make. Direct students to write their answers into the space provided on their copies of *Drawing Conclusions from the Faces of Cancer.*

Students should see that some people have a family history of cancer whereas other people do not. If students have difficulty expressing this idea, ask them whether the number of "yes" answers (the number of people who did have a family history of cancer) equals the total number of people who developed cancer (everyone in the class), and what this discrepancy means.

15. **Complete each row of the *Summary Profile* in turn, first asking teams to share their data with you, then totaling the data and entering them into the table. After you complete each row, give the teams time to discuss and agree on their conclusion and fill it into their worksheets.**

    - In the second row, students should see that the number of people who develop cancer increases with age (that is, the incidence of cancer increases with age). If students have difficulty expressing this idea, you may wish to ask a guiding question such as "What do you notice about the number of people who develop cancer in each life stage?" Encourage students to write their conclusion as a statement (for example, "The number of people who develop cancer increases with age.").

    - In the third row, students should see that cancer can develop in almost any tissue and organ in the body. They also may note that some types of cancer are more common than others.

      You may wish to ask students whether the fact that no one in this sample developed brain or uterine cancer means no one in the U.S. population gets this type of cancer. Students should recognize that this is not true. You also may wish to invite students to suggest other types of cancer that did not occur in this population and list them under "other" in the third row of the table.

    - In the fourth row, students should see that some people make choices or experience life events that increase their risk of developing cancer.

      Tip from the field test. Students may have difficulty distinguishing factors that increase risk for cancer from those that do not. If students express some uncertainty, ask them how they could find out about risk factors. You may wish to refer students to the Web site for the National Cancer Institute (http://www.nci.nih.gov) as an excellent source for current and reputable information about cancer.

16. **Ask whether anyone can suggest what the black dot on each person's set of cards might mean. Entertain several answers. If necessary, explain that these dots represent the period of life during which mutations may have occurred that eventually contributed to the development of cancer. Explain that students will learn more about these mutations in Activity 2. Ask the students to discuss in their teams what they notice about the dots.**

Give the students several minutes to look at the dots and discuss what they observe. If students seem to be confused about what they should be noticing, ask them guiding questions such as "What do you notice about the period of life in which each person's dot occurs and the period in which that person's cancer was detected?"

Be sure that students understand the difference between the period of life in which the mutations associated with the development of cancer occurred and the period in which the cancer was detected. In some cases, the dot appears in the same period of life that the cancer was detected. In most cases, however, the dot appears many years before the cancer was detected.

17. **Ask two or three teams to report what they observed about the dots and initiate a class discussion about the significance of these observations.**

    Help students understand that cancer develops across time and often many years intervene between the first cancerous changes and the symptoms that cause a person to seek medical help.

    As part of this discussion, you also may wish to ask students (1) what factors in people's lives improve their chance of recovering from cancer (for example, early detection and treatment); (2) what factors reduce their chances of early detection (for example, poor access to health care, either because of where they live or their socioeconomic status); and (3) what factors increase their chances of early detection (for example, participation in opportunities to be screened for cancer). Challenge students to support their answers by referring to specific people they learned about in this activity.

Collect and review the students' completed worksheets to assess their understanding of the activity's major concepts.

18. **Close the activity by asking students to complete the Discussion Questions on the bottom of *Drawing Conclusions* either in class or as homework. Briefly discuss their answers with them at the end of the period or at the beginning of the next.**

    Question 1 In this activity, all students in the class assumed the role of someone who developed cancer sometime in his or her lifetime. Is this an accurate representation of the risk of cancer among the American population? Explain your answer.

    No, this is not an accurate representation. Students should remember the opening exercise in which they learned that current statistics indicate that only 1 in 3 Americans develops cancer sometime in his or her lifetime. Point out that in this activity, students studied 30 people who *all* got cancer, but this does not mean that everyone will get cancer in his or her lifetime.

    Point out as well that students should not extrapolate from the *rates* of cancer illustrated in the activity. Although the general trends illustrated in these 30 people are accurate (for example, rates for lung, colon, and breast cancer are higher than rates for cervical, pancreatic,

and ovarian cancer), rates for other cancers are artificially exaggerated as a result of the small sample size. A striking example of this exaggeration occurs in the case of retinoblastoma. We included retinoblastoma to illustrate an example of a hereditary cancer, even though its incidence in the U.S. population is 1/12,000 to 20,000, not 1/30 as implied in this activity.

Question 2 What explanation can you offer for the observation you made about the incidence of cancer compared to age?

Answers will vary. Some students may suggest that it is related to the fact that cancer develops across time (which they learned when you discussed the black dots with them). Because older people have lived longer, they have a greater chance of developing it. Students will return to this question in Activity 3, *Cancer as a Multistep Process*.

Question 3 What is the most interesting or surprising thing you learned from this activity? What is the most important? Why?

Answers will vary.

Asking students to name the most important thing they learned challenges them to identify the activity's key ideas. If students have difficulty with this, ask questions based on the objectives in *At a Glance*.

Potential Extensions

Extend or enrich this activity by asking students to bring to class current newspaper or magazine articles about cancer. Display these in your classroom and, at the close of Activity 5, invite students to comment on them, drawing on what they learned about cancer during the preceding activities.

Activity 2

Cancer and the Cell Cycle

At a Glance

Focus: Students use five CD-ROM-based animations to help them construct an explanation for how cancer develops, then use their new understanding to explain several historical observations about agents that cause cancer.

Major Concepts: The growth and differentiation of cells in the body normally are precisely regulated; this regulation is fundamental to the orderly process of development that we observe across the life spans of multicellular organisms. Cancer develops due to the loss of growth control in cells. Loss of control occurs as a result of mutations in genes that are involved in cell cycle control.

Objectives: After completing this activity, students will

- understand that many different agents can cause cancer,
- understand that cancer represents a breakdown of the processes that regulate the growth of normal cells and tissues,
- recognize that cancer develops as a result of genetic damage that occurs to cells across time,
- be able to explain that cancer is associated with the occurrence of damage to particular classes of genes involved in the normal regulation of the cell cycle, and
- understand that studying the processes involved in the development of cancer has led to a significantly increased understanding of the normal cell cycle as well as to new strategies for treating cancer.

Prerequisite Knowledge: Students should be familiar with mitosis, the cell cycle, and terms such as "gene" and "mutation."

Basic Science-Public Health Connection: This activity focuses students' attention on how understanding the basic biology of cancer can help us make sense of the many observations people have made about risk factors related to cancer.

Introduction

Cancer has been described as a single disease and a hundred diseases. The description of cancer as a single disease arises from the observation that all cancers display uncontrolled growth, the ability to expand without limit. The description of cancer as a hundred diseases arises from the observation that cancer can appear as a result of different causes, in a variety of sites within the body, and that each type of cancer displays its own growth rate, prognosis, and treatability.

The discovery that all cancer involves a fundamental disruption in the growth of cells and tissues suggests that to understand cancer, we need to understand the events and processes that occur as both normal and abnormal cells grow and divide. In fact, much cancer research across the past two decades has focused on this challenge. This research has revealed a complex picture of how two classes of

genes, called proto-oncogenes and tumor suppressor genes, normally regulate the intricate sequence of cell cycle events. And it also has revealed how the accumulation of mutations in these genes can contribute to the development of an altered cell, a cell that has lost the normal controls on cell division.

In this activity, students gain a flavor of the initial confusion that existed among scientists about the causes of cancer by viewing several early accounts of possible relationships between the development of cancer and various internal and external factors. Students then use five CD-ROM-based animations to learn about evidence that helped scientists understand that (1) cancer involves the uncontrolled division of body cells; (2) cell division normally is precisely regulated; (3) cell cycle regulation is accomplished by two major types of genes; (4) cancer-causing agents often damage genes; and (5) when damage occurs to genes that regulate the cell cycle, the balance between signals that stimulate cell division and signals that inhibit cell division can change, leading the cell to divide more often than it normally would. As the activity closes, students use their new understanding of cancer to explain the relationships they learned about in Step 1.

Materials and Preparation

You will need to prepare the following materials before conducting this activity:

- Master 2.1, *Understanding Cancer* (make 1 copy per student)
- *Cell Biology and Cancer* CD-ROM (1 per team)

Follow the instructions on pages 28–29 to load the CD-ROMs on the computers students will use.

Note to teachers: If you do not have enough computers equipped with CD-ROM drives to conduct this activity, you can use the print-based alternative. To view and print the instructions and masters for this alternate activity, load the CD onto a computer and click the Print button on the main menu screen. The computer will display a screen showing the resources available for printing from the CD; click on the button labeled Non-CD Lesson Plan from the choices available for Activity 2, *Cancer and the Cell Cycle*. This will reveal the lesson plan and the masters for the alternate, non-CD-based activity. Click Print again to print these resources.

Procedure

1. **Introduce the activity by noting that people have wondered about the cause of cancer for thousands of years. Throughout this time, many correlations have been noted between the development of cancer and various internal and external factors. As examples of this, ask students to organize into their teams and view each of the *News Alert* videos on the CD-ROM. Distribute one copy of Master 2.1, *Understanding Cancer*, to each student and ask students to complete Section 1, Factors Reported to Be Associated with Cancer, by identifying**

   - **what each video suggests about the cause of cancer and**
   - **what evidence the video provides to support the claim.**

Divide the class into teams for this activity. The number of teams you will have depends on the number of computers equipped with CD-ROM drives you have available. We recommend that you ask students to organize into the same teams in which they worked in Activity 1, and place three of the students from one team at one computer and the other three students at a neighboring computer. This arrangement has the advantage that students who worked together in Activity 1 will work together or near one another in this activity as well.

Give the teams approximately 5 minutes to complete this task.

Notice that the videos describe reports of relationships between cancer and various causative agents that span more than 200 years. You may wish to draw students' attention to the length of time people have systematically studied the cause of cancer and also to the diversity of relationships that scientists studying the disease have identified and explained.

2. **Ask the students what each video suggests about the cause of cancer and what evidence was provided to support the claim.**

   To increase the level of student participation, ask one team to describe what a particular video suggests about the cause of cancer and a different team to describe the evidence on which this claim was based.

   At the close of the reporting, you may wish to ask students whether the evidence presented in these videos is convincing and why. This is a good point in the activity to remind students of the difference between correlation and causation and ask what type of evidence would demonstrate causation.

3. **Explain that each news item describes what has proven to be a real relationship between the development of cancer and the factor described. Ask students what general question all four videos raise when they are considered collectively.**

   Students may suggest several questions that could be asked about the videos. Help students see that the fundamental challenge facing scientists interested in understanding cancer was to explain how so many diverse factors can cause it. Students may phrase this question as "How can so many different factors all cause cancer?" or "What does each of these factors do to cause cancer?"

   Tip from the field test. Students may ask questions that relate more to the medical aspects of cancer than to its underlying cause. If students are having difficulty recognizing the question that these four videos raise about cancer's cause, you may wish to rephrase the question as "What do you think may have confused researchers trying to understand what goes wrong in cancer cells?" or "The number of different agents that can cause cancer was one of the most confusing aspects of cancer

to early researchers. Why was this confusing?" or "What do you think all these agents had in common and why was it important to discover that?"

4. **Explain that research across the past 30 years has helped scientists understand how so many different factors can cause cancer. Explain that next students will view five CD-ROM-based animations that will help them construct an explanation of the cause of cancer. They then will use their understanding of cancer's cause to explain the relationships described in the *News Alert* videos.**

Students may be surprised to learn about the cell cycle in an activity that focuses on risk factors for cancer. Point out that understanding disease typically requires scientists to examine basic cellular processes, and that understanding those processes can, in turn, help health care workers develop better prevention and treatment strategies.

5. **Direct the students to view the animations on the CD-ROM. Then ask them to complete Section 2, Building an Explanation for the Cause of Cancer (on *Understanding Cancer*) by writing a one-sentence statement that summarizes what they learned from each animation.**

   - *Animation 1* (the animation of abnormal cell growth) should lead students to conclude that **cancer involves the uncontrolled division of body cells**.
   - *Animation 2* (the introduction to the cell cycle) should lead students to conclude that **cell division normally is precisely regulated**.
   - *Animation 3* (the information on proto-oncogenes and tumor suppressor genes) should lead students to conclude that **cell cycle regulation is accomplished by two major types of genes**.
   - *Animation 4* (the information on the mutagenicity of carcinogens) should lead students to conclude that **cancer-causing agents often damage genes**.
   - *Animation 5* (the information on the effect of damaging cell cycle genes) should lead students to conclude that **when damage occurs to genes that regulate the cell cycle, the balance between signals that stimulate cell division and signals that inhibit cell division can change, leading the cell to divide more often than it normally would**.

Steps 6–8 represent the closure steps for this activity. Step 6, in particular, focuses students' attention on the activity's major concepts.

6. **After the students have completed Section 2 on *Understanding Cancer*, point out that their five statements constitute a basic explanation of what goes wrong when a cell becomes cancerous. Ask one or more teams to read their statements to the class, then invite clarifying comments and questions from the rest of the students.**

7. **Ask the teams to complete Section 3, Explaining Factors Associated with Cancer, on *Understanding Cancer* by reviewing the information in Section 1 and writing four one-sentence explanations for how the relationship each video describes can be understood in the light of what scientists know today about the cause of cancer.**

   Give students approximately 5 minutes for this task, then ask a spokesperson from each team to explain one of the videos.

   Students may have difficulty with this step, primarily because they lack sufficient background in biology to make the connections required to

explain "causative" agents of cancer. For this reason, we suggest that you ask your students to provide only the most basic explanations, such as those provided in bold type below. After they have done so, you can explain as much of the detail as you think is appropriate and will be interesting to the class.

- *News Alert! Cancer and Chemical Poisons.* Students should be able to suggest that **a chemical in the coal dust caused damage to genes that regulate the cell cycle.**

  Pott was probably the first person to associate a specific type of cancer (scrotal cancer) with a specific occupation (chimney sweeping). Pott believed the problem was the coal soot that caught in the skin folds of the scrotum. In 1918, coal tar was shown to cause skin cancer in rabbits, and in 1924 the causative agent was identified as polycyclic aromatic hydrocarbons, especially benzo (a) pyrene.

- *News Alert! Cancer and Your Family History.* Students should be able to suggest that **children with inherited retinoblastoma have inherited an error (mutation) in a gene that regulates the cell cycle.**

  Retinoblastoma, a relatively rare cancer, is a highly malignant tumor of the eye. If left untreated, the malignancy moves from the eye along the optic nerve to the brain, from where it metastasizes to other tissues. Slightly more than one-third of retinoblastoma cases are inherited. The remaining cases are sporadic (not inherited). The age of onset of the inherited type is approximately 10 months, on average 8 months earlier than the sporadic type. Tumors of both eyes occur only with the inherited type.

  A mutation or deletion in the long arm of chromosome 13 is associated with the development of retinoblastoma. Both alleles of the gene involved, the *RB* gene, are either missing or altered in nearly every case of retinoblastoma (whether inherited or sporadic). The gene's normal product has an inhibitory effect on cell division.

  Children who inherit an altered allele of the *RB* gene are heterozygous for the chromosome 13 abnormality. They are at high risk for developing retinoblastoma because only a single mutation or deletion of the normal *RB* gene will result in a cell initiating uncontrolled cell division. The mutation rate for this gene is high enough that there is significant risk of experiencing the mutation in the cells of both eyes (thus, the risk of developing retinoblastoma in both eyes in the inherited type).

  In sporadic (nonhereditary) retinoblastoma, both alleles of the *RB* gene are normal, and each one must be mutated in the same cell for the tumor to arise. In contrast with hereditary retinoblastoma, the likelihood of this occurring in both eyes is so low that for all practical purposes, it does not occur.

- *News Alert! Cancer and Radiation Exposure.* Students should be able to suggest that **exposure to X-rays damages genes that regulate the cell cycle.**

Ionizing radiation is a well-known human carcinogen. The first reports of association between X-rays and cancer appear in the literature in the early 1900s. Subsequent reports include the association between radium exposure and leukemia (for example, Marie Curie died of leukemia); radium exposure and osteosarcomas (for example, cancer developed among painters of luminescent dials in watch factories in the 1930s); and radiation from nuclear tests and cancer (for example, children in the Marshall Islands exposed to radioactive iodine released from a nuclear test displayed a significant increase in thyroid cancer).

Carcinogenesis from ionizing radiation is believed to occur through the formation of mutagenic oxygen free radicals. Ionizing radiation is clearly carcinogenic when presented at unusually high doses, but it has been difficult to quantify its effect when presented at low doses. Because the assumption is that any amount of exposure has some effect, federal regulations mandate that exposure to radiation be kept "as low as reasonably achievable."

Steps 6 and 7 provide excellent opportunities to assess students' understanding of the activity's major concepts. In Step 6, students should be able to express five key ideas about the regulation of cell division, and in Step 7, they should be able to apply this understanding to explain how certain risk factors increase a person's chance of developing cancer.

- *News Alert! Cancer and UV Light.* Students should be able to suggest that **exposure to UV light damages genes that regulate the cell cycle.**

  The relationship between sun exposure and skin cancer has been clarified greatly across the past century. In the late 1800s, observers noticed that sailors exposed to the sun developed a variety of abnormal lesions called "sailor's skin," and in the early 1900s, an increased risk of skin cancer was observed among farmers. By 1928, researchers had demonstrated the carcinogenic effect of UV radiation on the skin of laboratory animals. Today, scientists recognize excessive exposure to UV radiation (whether from the sun or other sources) as a key risk factor for skin cancer.

8. **Close the activity by asking students what the activity reveals about science's ability to bring order to even the most bewildering set of observations.**

   Students should recognize that science helps us explain and relate observations that we make about the natural world. You may wish to ask students if they can think of other examples of observations that have been organized and made comprehensible through the work of science. Students may propose the atomic theory, the cell theory, and the germ theory of disease as important organizing explanations in science. If they do not mention evolution, point out that evolution is the most important organizing explanation in biology.

Activity 3

Cancer as a Multistep Process

At a Glance

Focus: Students use random number tables and a CD-ROM-based simulation to test several hypotheses about the development of cancer.

Major Concepts: No single event is enough to turn a cell into a cancerous cell. Instead, it seems that the accumulation of damage to a number of genes ("multiple hits") across time leads to cancer.

Objectives: After completing this activity, students will

- understand that cancer results from the accumulation of genetic damage to cells across time and
- be able to explain the increase in cancer incidence that occurs with an increase in age in terms of a multiple hit (mutations in a number of genes) hypothesis for cancer's development.

Prerequisite Knowledge: Students should be familiar with the concepts taught in Activities 1 and 2. Students also should have a basic knowledge of probability. The annotation to Step 6 describes a short exercise you can do with students to remind them of the laws of probability.

Basic Science-Public Health Connection: This activity highlights the contribution epidemiology has made to our understanding of cancer. Students discover how determining and analyzing the frequencies with which cancer occurs in large populations has provided compelling though indirect evidence that human cancer is a multistep process. Students then consider the implications of this understanding of cancer for personal and public health.

Introduction

The process by which a normal cell is transformed into a malignant cell involves many changes. Cancer cells display a host of striking differences from their normal counterparts, such as shape changes, changes in their dependence on growth factors, and a multitude of biochemical differences.

One of the earliest questions scientists asked about these phenotypic differences was how they are generated. Another question was whether these differences arise all at once, at a moment when the cell experiences a sudden, catastrophic shift from "normal" to "malignant," or gradually, across time, as a result of many small events, each contributing yet another characteristic to a set that in sum gives the cell a malignant phenotype.

In this activity, students examine some of the epidemiologic data that suggest that the development of cancer is a multistep process. Students study a graph of colon cancer incidence by age, answer an initial set of questions about the relative risk of developing colon cancer at various ages, and propose answers to

the question of why this risk increases with age. Students then use random number tables and a CD-ROM-based simulation to test several hypotheses about the development of colon cancer (for example, colon cancer develops as a result of a single event within a cell, colon cancer develops as a result of two independent events within a cell, and so on). Finally, students use their understanding of the development of cancer as a multistep process to explain (1) the increased incidence of cancer with age, (2) the development of cancer decades after exposure to known carcinogens, and (3) the increased incidence of cancer among people with inherited predispositions.

Materials and Preparation

You will need to prepare the following materials before conducting this activity:

- Master 3.1, *Colon Cancer Incidence by Age* (make 1 copy per student and 1 transparency)
- Master 3.2, *Random Number Tables* (make enough copies to have 1 data set per student)

  Copy the master and cut out one data set for each student in your class.
- Master 3.3, *Collecting the Data* (make 1 transparency)
- Master 3.4, *Graphing the Data* (make 1 copy per student and 1 transparency)
- Master 3.5, *Using the Hit Simulator* (make 1 copy per student)
- Master 3.6, *Testing an Explanation by Looking at Additional Data* (make 1 copy per student)
- coins (1 penny, 1 nickel, and 1 dime for each student, only if you plan to conduct the review of probability described in Step 6)
- a hat (or other container) with folded slips of paper containing the numbers from 1 to 25
- *Cell Biology and Cancer* CD-ROM

Follow the instructions on pages 28–29 to load the CD-ROMs on the computers the students will use.

Note to teachers: If you do not have enough computers equipped with CD-ROM drives to conduct this activity, you can use the print-based alternative. To view and print the instructions and masters for this alternate activity, load the CD onto a computer and click the Print button on the main menu screen. The computer will display a screen showing the resources available for printing from the CD; click on the button labeled Non-CD Lesson Plan from the choices available for Activity 3, *Cancer as a Multistep Process*. This will reveal the lesson plan and the masters for the alternate, non-CD-based lesson. Click Print again to print these resources.

Procedure

1. **Open the activity by reminding students of the increase in cancer incidence with age that they observed in Activity 1. Explain that in this activity, they will investigate the biological basis for this increase.**

   It is very important to set this activity in the context of Activities 1 and 2.

Without this context, students may complete this activity "by rote" and never see how it relates to our growing understanding of the biological basis of cancer.

2. **Distribute one copy of Master 3.1, *Colon Cancer Incidence by Age*, to each student and ask the students to work in pairs to answer the questions below the graph.**

   Give students about 5 minutes to complete this task.

3. **Display a transparency made from *Colon Cancer Incidence* and invite the students to share their answers to the questions.**

   Question 1 How likely is it that you will develop colon cancer this year?

   Students should answer that the risk is so low that they cannot read it from the graph. You may wish to ask whether children under 15 ever develop colon cancer. In fact, those few children who do have genetic conditions that predispose them to the development of cancer.

   Question 2 How likely is it that someone who is 60 years old will develop colon cancer this year?

   The risk is significantly higher (approximately 70 per 100,000 persons).

   Question 3 How likely is it that someone who is 80 years old will develop colon cancer this year?

   This risk is even higher (about 350 per 100,000 persons).

   Question 4 How can we explain this change in the risk of a person developing colon cancer?

   Answers will vary. Students may suggest that as people age, they become more susceptible to cancer. Some may also suggest that it takes time for the mutations involved in the development of cancer to accumulate. Accept all reasonable answers, explaining that in this activity, students will have a chance to test a possible answer to this question.

4. **Circle the last question on the transparency or write it on the board and point out that this question is the central issue in this activity. Explain that many years ago epidemiologists recognized that this change in cancer risk provided an important clue about the cause of cancer. This activity challenges students to retrace the thinking of these scientists and discover this clue for themselves.**

   If students are unfamiliar with the term "epidemiology," explain that it is the study of the incidence of disease in a population.

5. **Remind students that one way scientists answer questions is by developing and testing hypotheses, or tentative explanations. For example, one explanation that might be offered for the development of cancer might be summarized as "One mutation in a certain gene in a cell causes that cell to become**

cancerous" (the one-hit hypothesis). Another explanation might be summarized as "Two mutations in separate genes of a cell are required before the cell becomes cancerous" (the two-hit hypothesis), and so on. Ask students if they can tell by looking at the colon cancer graph which of these two explanations for the development of cancer best explains the data.

Students likely will answer that they cannot tell just by looking at the graph.

6. **Explain further that scientists often use models to test their explanations. In this activity, students will use two simple models, one involving random number tables and the other using a simulation on the CD-ROM, to test several alternate explanations for the development of cancer.**

   If your students are not familiar with some of the basic concepts of probability, you may wish to conduct the following short exercise:

   Give each student a penny, a nickel, and a dime, and ask students to toss each coin one time and leave the coins lying on their desks where they landed. Ask the students to raise their hands if they got a "heads" on their penny. Count the number of students who raise their hands and point out that this represents about 50 percent of the class. Then, ask students to indicate how many got heads on both their penny and their nickel. Again, count the number of students who raise their hands and point out that this value is close to 25 percent of the class. Finally, ask students to raise their hands only if they got a heads on all three of their coins (the penny, the nickel, and the dime). This number should be about one-eighth of the class. Ask students what pattern they see in these data. Students should see that the probability of independent events happening together is lower than each event's individual probability. Use your judgment to decide whether to explain to students how to calculate the probability of such occurrences (for example, the probability of getting heads on three coins tossed individually is $^1/_2 \times {}^1/_2 \times {}^1/_2 = {}^1/_8$).

7. **Distribute one data set from Master 3.2, *Random Number Tables*, to each student and explain that students will use these data to understand the implications of the following two hypotheses for the incidence of cancer in a population (the class):**

   1. **Cancer develops as a result of a single mutation (one-hit hypothesis).**
   2. **Cancer develops as a result of two independent mutations (two-hit hypothesis).**

8. **Explain that the data sets the students hold are called random number tables and were made as a computer randomly chose numbers between 1 and 25 to correspond with the students' imagined life spans. Explain that the first column on the table represents the students' ages, and that the second and third columns on the table represent the numbers the computer chose.**

9. **Conduct the following exercise:**

   - **Ask a student to draw a number out of the hat and announce the number to the class. Write the number on the board.**

     For example, imagine that the student drew the number 10.

   - **Explain that this number represents a mutation in gene 1. Ask students to examine the column labeled "Gene 1" on their random number table to determine whether they have the number chosen. If they do, they should circle it and note the age at which it occurred.**

     Students should look down the column labeled "Gene 1" for the first occurrence of the "unlucky" number (in this example, 10). If the number occurs more than once, they should ignore the second (and any subsequent) occurrence.

   - **Ask another student to draw a number out of the hat and announce it to the class. Write the number on the board.**

     For example, imagine that the student drew the number 4.

   - **Explain that this second number represents a mutation in gene 2. Ask students to examine the column labeled "Gene 2" on their random number table to determine whether they have the second number chosen. If they do, they should circle it and note the age at which it occurred.**

     Students should look down the column labeled "Gene 2" for the first occurrence of the "unlucky" number (in this example, 4). If the number occurs more than once, they should ignore the second (and any subsequent) occurrence.

10. **Display a transparency made from Master 3.3, *Collecting the Data*, and explain that you are going to use this table to tally the number of people in the class who would have developed cancer at each age if the one-hit or two-hit hypotheses for the development of cancer were true.**

    Explain that to discover the number of people who would have developed cancer, the students need to examine their random number tables according to the following instructions:

    - **Tell students that first the class is going to approximate what might happen if the one-hit hypothesis were true (that is, if one mutation were sufficient to cause a normal cell to become cancerous). Ask students to imagine that if they found the first "unlucky" number in the column labeled "Gene 1," it meant a gene in one of their cells experienced a cancer-causing mutation. Explain that if the one-hit hypothesis were correct, the age at which the unlucky number *first* appears in the column labeled "Gene 1" would be the age at which they developed colon cancer.**

Note that some students likely will not encounter the unlucky number and, therefore, will not develop cancer.

- **Poll the class to determine how many students developed cancer under this model at each age (5–100 years) and fill this information into the column labeled "Number of People Who Developed Cancer—One Hit" on the transparency.**

  Only students who had the first unlucky number (in the example, 10) in the column labeled "Gene 1" should indicate that they developed cancer.

- **Next, tell students that the class is going to approximate what might happen if the two-hit hypothesis were true (that is, if two mutations were required to cause a normal cell to become cancerous). Ask students to imagine that if they encountered the second "unlucky" number in the column labeled "Gene 2," it meant they experienced a cancer-causing mutation in another gene in the same cell that experienced the first mutation. Explain that the age at which the second unlucky number first appears in the column "Gene 2" is the age at which they experienced this second mutation. However, if the two-hit hypothesis were correct, they did not develop colon cancer until the age at which they encountered *both* unlucky numbers (the unlucky number in the column labeled "Gene 1" *and* the unlucky number in the column labeled "Gene 2").**

  Tell students that it does not matter in what order the mutations occur. Emphasize that in order to have developed cancer under this model, they would need to have both the first unlucky number in the column labeled "Gene 1" *and* the second unlucky number in the column labeled "Gene 2" (in the example, both a 10 in the first column and a 4 in the second column). The age at which they developed cancer is the age by which they had experienced *both* mutations.

  Note that many students likely will not encounter both unlucky numbers and, therefore, will not develop cancer.

- **Poll the class to determine how many students developed cancer at each age under this second model and display the data in the appropriate column on the transparency.**

- **Work with the class to accumulate the running total of cancer cases observed in the class population at each age under the one-hit and two-hit models and write these values into the last two columns on the transparency.**

  These running totals represent the cumulative number of people who developed colon cancer at or before each age.

11. **Ask students whether they see any pattern in the incidence of cancer in this population (the class).**

Students may see that fewer people developed cancer under the second model (the two-hit hypothesis) than under the first (the one-hit hypothesis) and that those who did develop cancer under the two-hit hypothesis tended to do so later in life. Encourage students to express this observation as a generalization (for example, the chance, or "risk," of developing cancer early in life diminished as the number of events involved in its development increased). If students are able to recognize this, ask them what this observation suggests about the development of cancer. *Do not give students this answer—that it suggests that more than one event may be involved in the development of cancer—but indicate that they should think about this question as they complete the activity.*

12. **Distribute one copy of Master 3.4, *Graphing the Data*, to each student and direct students to work in pairs to construct two graphs that illustrate the chances that a person in this class would have developed colon cancer by a certain age if the one-hit or two-hit hypothesis were true.**

13. **Ask students to examine the graphs they generated in Step 12 and decide which hypothesis best fits the actual data on the incidence of colon cancer.**

    Students likely already recognize that fewer cancer cases were encountered when two mutations (two hits) were required. However, they also may see from the graph that neither hypothesis fits the colon cancer data well.

14. **Ask students to predict what the results of a simulation such as this might be if three, four, or as many as five mutations (five hits) must occur prior to the onset of cancer.**

    Students may be able to suggest that cancer would become an increasingly rare event and would tend to occur later and later in life as the number of mutations required for its development increases.

15. **Distribute one copy of Master 3.5, *Using the Hit Simulator*, to each student. Direct students to organize into their teams and follow the instructions provided to use the CD-ROM-based simulation to test their predictions and decide what type of model for the development of colon cancer best fits its observed incidence.**

    This simulation is a sophisticated tool that students can use to observe how mutation frequency and the number of mutations (hits) required for the development of cancer affect the incidence of cancer in a population. Although students can simply experiment with the simulation, their experience likely will be more meaningful if they follow the guidelines provided on *Using the Hit Simulator*.

    Give students approximately 20 minutes to learn to use the simulation, test their predictions, and answer the questions on *Using the Hit Simulator*.

These questions focus students' attention on the activity's major concepts. Encourage students to express their understanding of cancer using the language of cells and genes.

16. Convene a class discussion in which you invite students to share their answers to the questions on *Using the Hit Simulator.*

- **Investigate the Effect of Changing the Number of Hits Required**

Question 1 How does the incidence of cancer change as you require a greater number of hits for a cell to become cancerous?

Students should see that the greater the number of hits required, the fewer the number of people who develop cancer and the later in life they tend to develop it. Students may note that with the number of hits set at 1 and the mutation rate set at 0.5 (50 percent), nearly everyone in the population gets cancer by age 25. As they increase the number of hits required, the curve shifts to the right (people get cancer later in life), though most people still develop it eventually.

Question 2 Recall the graph of the incidence of colon cancer that you observed at the beginning of this activity. Did the incidence of cancer in any of the runs you just completed match the incidence of cancer recorded in that graph? Explain your answer.

Students should recognize that none of the runs matched the actual incidence of colon cancer. You may wish to remind students that they learned in Activity 1 that only about 1 in 3 people in the United States develops cancer sometime in his or her life.

Question 3 What can you conclude from this observation?

Students should see that because the curve shifted to the right (toward the development of cancer later in life) as more hits were required, the results suggest that more than 1 hit likely is involved in the development of cancer. Astute students also may say that because almost everyone eventually developed cancer in these simulations, the mutation rate of 0.5 (50 percent) likely is too high.

- **Investigate the Effect of Changing the Mutation Rate**

Question 4 How does the incidence of cancer change as the mutation rate increases?

The incidence of cancer increases as the mutation rate increases. Students should see that with the simulator set at a mutation rate of 0.1 (10 percent), a smaller proportion of the population develops cancer than when the simulator is set at 0.5 (50 percent). With the simulator set at a mutation rate of 1 (100 percent), everyone gets cancer between the ages of 0 and 5.

Question 5 Recall the graph of the incidence of colon cancer that you observed at the beginning of this activity. Did the incidence of cancer in any of the runs you just completed match the incidence of cancer recorded in that graph? Explain your answer.

Students should recognize that none of the runs matched the actual incidence of colon cancer. They should recognize, however, that the curve(s) generated with the mutation rate set at 0.1 or lower was/were more in line with the observed incidence than the curves generated with higher mutation rates.

Question 6 What can you conclude from this observation?

Students should recognize that these results suggest that the actual mutation rate is somewhat lower than 0.5 (50 percent), and maybe even lower than 0.1 (10 percent).

- **Investigate the Effect of Changing Both the Number of Hits Required and the Mutation Rate**

Question 7 What can you conclude from your observations?

Students should see that the curves generated by some of these runs begin to resemble the incidence of colon cancer observed on the graph they examined at the beginning of the activity. Encourage students to suggest combinations of numbers of hits and mutation rates that seem to give realistic results, but caution students not to use this simulator (which was designed for educational purposes*, not research) to try to make an absolute determination of number of hits and mutation rate.

You may wish to point out that a mutation rate of 0.04 is the same rate that was used in the random number table exercise. Challenge students to demonstrate this by comparing the graphs they made of the one-hit and two-hit hypotheses with the curves generated by the simulator. Note that using the CD-ROM-based simulator allows them now to test the predictions they made in Step 14.

*Note that the graph of the incidence of colon cancer used in Step 2 is actually the number of people in a population of 100,000 who will be diagnosed with colon cancer at each age. The graphs students created in class and the graphs generated by the *Hit Simulator* are somewhat different because they plot cumulative numbers (the total number of people who will have developed colon cancer at or before that age).

- **Summary**

Question 8 What clue did the change in risk of colon cancer provide scientists about the cause of cancer?

Students should be able to explain that the increase with age in colon cancer incidence suggested to epidemiologists that more than one mutational event was involved in cancer's development. Similar graphs of age-dependent cancer incidence, plotted for many other

Whereas Activity 2 illustrates the contribution that cell biologists and geneticists have made to understanding cancer, Activity 3 illustrates the contribution that epidemiologists have made. One of the most exciting aspects of cancer research in recent years has been the construction of an understanding of cancer that unifies the work of many types of scientists studying cancer for more than 100 years.

types of adult cancer, provided additional support for the hypothesis of multistep carcinogenesis. In fact, one of the goals of research today is to identify each of the steps and genes involved in the long and complex succession of events that occurs to create the malignant growth of cancer cells.

Note that this question returns students to the challenge they were given in Step 4.

17. **Challenge your students to evaluate the models they used to test the different hypotheses for the development of cancer (that is, to think about the ways in which the random number table exercise and the CD-ROM-based simulation do and do not match reality).**

    Remind students that all models are inaccurate in some respects. For example, the mutational events within cells may not be completely random, as the models assume. The models also assume that the probability of each individual mutational event is the same, and this may not be the case. There is some evidence, for example, that some mutations increase the probability that other mutations will occur. In addition, the models do not consider that some mutations may be detected and repaired. Nevertheless, the fact that the models the students used are not perfect does not mean they are not useful tools for understanding how disease processes work.

18. **Close the activity by distributing one copy of Master 3.6, *Testing an Explanation by Looking at Additional Data*. Ask students to use their understanding of cancer as a multistep process to explain each of the observations listed.**

    Question 1 Cancer is a disease of aging.

    Students should be able to explain it takes time for all of the mutations involved in the development of cancer to accumulate, and that this explains why the incidence of cancer increases with age (that is, why cancer is more likely to "strike" in the middle or later years than in childhood, youth, or young adulthood.

    Question 2 You've come a long way, baby.

    Students should be able to explain that as these women began to smoke, they began to accumulate cancer-causing mutations in their lung cells. Because the accumulation of these mutations to the point where a cell becomes cancerous takes time, the results of the increase in the number of women smoking (in the form of an increase in lung cancer among women) did not begin to appear for 20 to 25 years.

    Question 3 Genes and increased susceptibility.

    Students should recognize that if a person is born with a cancer-causing mutation already present in his or her cells, he or she has

The questions on *Testing an Explanation* are challenging, but they represent an excellent opportunity for you to evaluate your students' understanding of the activity's major concepts and their ability to apply their understanding to novel situations.

already experienced the first step toward the development of cancer and, thus, has a higher risk of accumulating all of the mutations required for the development of cancer than a person who does not carry such a mutation.

Activity 4

Evaluating Claims About Cancer

At a Glance

Focus: Students identify claims about UV exposure presented in a selection of media items, then design, execute, and report the results of an experiment designed to test one such claim.

Major Concepts: Scientists use systematic and rigorous criteria to evaluate claims about factors associated with cancer. Consumers can evaluate such claims by applying criteria related to the source, certainty, and reasonableness of the supporting information.

Objectives: After completing this activity, students will

- understand that many people and organizations make claims about factors associated with the development of cancer and about agents that may help prevent or cure cancer;
- be able to explain that scientists evaluate such claims by applying systematic and rigorous criteria;
- be able to apply criteria such as the source, certainty, and reasonableness of the supporting information to media claims about cancer; and
- recognize that understanding the biology of cancer and the nature of science can help individuals and society make reasoned choices about factors related to cancer.

Prerequisite Knowledge: Students should have sufficient understanding of the methods of science to develop a hypothesis, design an experiment to test that hypothesis, and draw reasonable conclusions from the results.

Basic Science-Public Health Connection: This activity provides the opportunity for students to discover how science can help individuals and society evaluate claims about cancer.

Introduction

Although most of your students will never acquire or need a detailed understanding of the biology of cancer, all of them will need to understand and evaluate claims about cancer that they encounter in casual conversation, in media items, and even in reports from health care workers. Some of these claims may be vague, either in substance or in origin. Some may be exciting and seem to offer great hope. Others may be alarming. In some cases, these claims may conflict.

How do scientists evaluate claims about cancer? And how can your students, as scientifically literate citizens, evaluate such claims, both for their own satisfaction and as a solid foundation for thinking about and voting on policy issues?

In this activity, students examine several media items about exposure to the sun and the development of skin cancer and work in teams to identify the claims

that each media item makes. Students are challenged to describe ways that scientists might evaluate these claims, and then are introduced to a model system involving UV (ultraviolet light)-sensitive yeast that they can use to test aspects of these claims. Each team designs, executes, and presents the results of a controlled experiment testing one of these aspects. The activity ends with a class discussion of (1) how scientists evaluate claims about cancer and (2) the criteria that nonscientists can use to evaluate such claims.

Materials and Preparation

You will need to prepare materials in advance for the laboratory exercise. Ordering information and preparation directions are on page 70, immediately following the activity.

You will need to prepare the following additional materials before conducting this activity:

- Master 4.1, *Media Item 1* (make enough for one-fourth of the class)*
- Master 4.2, *Media Item 2* (make enough for one-fourth of the class)*
- Master 4.3, *Media Item 3* (make enough for one-fourth of the class)*
- Master 4.4, *Media Item 4* (make enough for one-fourth of the class)*
- Master 4.5, *Using a Model System to Test Claims About UV Light* (make 1 copy per student)
- Master 4.6, *Evaluating Claims About Cancer* (make 1 copy per student)

*You will need one *Media Item* handout for each student in your class. Note that every student in one team gets the same handout, but different teams get different handouts.

Procedure

DAY 1

1. **Ask students to organize into teams. Distribute the masters so that each team has a different master; each member of a particular team should have the same master.**

2. **Direct students to read their media items, then work together to identify the major claims that their assigned item makes about the product, ultraviolet (UV) light, and cancer. Ask students also to describe the evidence on which these claims seem to be based.**

   Give the teams about 5 minutes for this discussion.

3. **Conduct a brief class discussion about the media items by asking the following questions:**

   - **What claims did you find in the media items? What evidence did the items provide to support these claims?**

     Students should not find it difficult to identify these claims. Possible answers include the following:

- *Media Item 1*: A new sunscreen gives 10 times more protection against sunburn than others, but still allows tanning.
- *Media Item 2*: A new brand of sunglasses protects eyes against UV light.
- *Media Item 3*: Cellophane protects against sunburn but still allows tanning.
- *Media Item 4*: Some clothing offers higher protection from UV light than other clothing.

The media items do not provide any evidence to support these claims.

- **What claims about such products and/or cancer have you heard during your lifetime? From whom (and where) do you hear such claims?**

  Students likely have heard many claims. Allow them to list not only outlandish claims that they may have heard in the media, but also more reasonable claims that they may have heard from parents, friends, and even reputable magazines and health care professionals. Technically, any information that we hear or read about cancer is a "claim" that someone is making.

- **How do scientists evaluate such claims?**

  Students should already understand that scientists evaluate such claims through rigorous experimentation, the requirement of evidence to support a claim, careful review by other scientists of procedures and conclusions, and the requirement that results be replicable. Look for these and similar answers from your students; if they are not forthcoming, ask probing questions such as "Is it sufficient for a scientist to make a claim without providing evidence to back it up?" and "If certain results can be obtained only by one scientist working in a particular laboratory, what would you think of claims based on these results?"

4. **Explain that in this activity, students will have an opportunity to test claims that are similar to those they encountered in their media items and will learn questions that citizens can ask about claims they hear in the popular press and from other sources.**

5. **Distribute one copy of Master 4.5, *Using a Model System to Test Claims About UV Light*, to each student. Ask students to work in their teams to design and conduct a controlled experiment that tests a claim related to their media item.**

   You may wish to explain that often scientists use model systems to evaluate certain claims that would not be appropriate to test using people as subjects. Often, these model systems involve other species. In this activity, students will use yeast as their model system.

Students have the opportunity here to experience how scientific experiments can lead to reasonable claims about how individuals can help prevent skin cancer. Point out that basic experiments, such as the one they are about to conduct, have led to a variety of actions on behalf of public health, including the banning of certain food additives and warnings they see on consumer products.

Circulate through the room as students read the information provided on *Using a Model System* and begin to design and execute their experiments. Notice that students will not be able to test the actual claims their media items make, but should be able to test related claims. You may have to ask probing questions to help students see how to use the yeast to test these claims. Following are suggestions for possible experiments:

- *Media Item 1*: Students can test the relative abilities of different brands of sunscreen or different SPF values to protect the yeast from UV light.
- *Media Item 2*: Students can test the relative abilities of different brands and types of sunglasses to protect the yeast from UV light.
- *Media Item 3*: Students can test the relative abilities of different colors of cellophane to protect the yeast from UV light.
- *Media Item 4*: Students can test the relative abilities of different colors and thicknesses of cloth to protect the yeast from UV light.

Tip from the field test. Although students will only be able to test certain aspects of the claims each media item makes, you may wish to challenge your students to identify what part(s) of the claim they are *not* testing and to describe how they might test those parts of the claim.

6. **Conclude Day 1 by asking each team to describe to the class the claim they are testing and the method they are using to test the claim.**

DAY 2

1. **Direct students to collect the plates from their experiments and record their results. Then convene a class discussion and ask each team to report briefly on its experiment. As each team reports, ask students what additional information they would need to be able to answer the experimental question more completely or to be able to apply their findings to humans.**

   Students should follow the outline provided in *Using a Model System* as they report on their experiments.

2. **Acknowledge the value of scientific research in evaluating claims, then ask students how nonscientists can evaluate similar claims that they encounter in the media or from other sources. List their ideas on the board or a transparency.**

   Students should recognize that they do not have the expertise, equipment, or time to experimentally evaluate each claim that they hear about cancer, but they can carefully consider claims to determine their source, whether they are supported by evidence, and how reasonable they are (that is, whether they seem to fit within existing knowledge or seem outlandish). Sometimes "outlandish" claims are correct, but this usually is not the case, and students should understand this.

3. **Challenge each team to use the results of its own experiment to develop a media item similar to the item they used in Step 1. Point out that this item does not need to sell something but can be designed to inform the public about the results of their work. Remind teams to use the list the class generated in Step 2 as a guide for writing credible claims.**

Collect teams' media items here, or after they refine the items based on feedback they receive in Step 5, to assess students' understanding that credible claims are supported by evidence.

4. **Distribute one copy of Master 4.6, *Evaluating Claims About Cancer*, to each student. Explain that this worksheet provides a set of questions that can help the scientifically literate citizen evaluate claims about science and health. Ask teams to exchange the media items they developed in Step 3 and to evaluate them by answering the questions on *Evaluating Claims*.**

5. **Invite partner teams to meet and share their analyses of their media items.**

   Remind students that useful feedback identifies both good features and features that need to be refined, and also provides specific suggestions for refinement.

   Give students about 5 minutes for this discussion. Circulate among the teams during their discussions and ask questions or make suggestions as appropriate. You may wish to challenge teams to revise their media items based on the feedback they receive.

6. **Close the activity by asking students how understanding the biology of cancer and the nature of science can help individuals and society make reasoned choices about factors related to cancer.**

   Students should be able to explain that scientific research identifies risk factors for cancer (for example, UV radiation) and also develops and tests products designed to protect against cancer (for example, sunscreen). Students should also recognize that understanding how scientists test claims and that being familiar with the requirements of evidence can help scientifically literate citizens evaluate claims they hear about cancer.

Asking students how science helps people make choices about factors related to cancer leads them back to one of the activity's major concepts.

Potential Extensions

Invite students to bring in samples of other media items, including Web-based advertisements, for the class to evaluate using the criteria developed in Day 2, Step 2 and the questions on Master 4.6, *Evaluating Claims About Cancer*.

Laboratory Preparation

1. *Four weeks before conducting the laboratory exercise.* Order the following from Carolina Biological Supply:

   a. YED yeast strain G948-IC/U, catalog # CD-17-3634
   b. YED agar medium, catalog # CB-17-3650

   Allow two weeks for delivery. Carolina Biological Supply will only ship live materials on Mondays, Tuesdays, and Wednesdays.

2. *Up to one week before conducting the laboratory exercise.* Prepare petri plates containing YED agar medium following the directions on the package. You will need 1 plate per student plus 1 additional plate per team; we recommend preparing extra plates to allow for mistakes and contamination.

   Prepare the plates up to 1 week in advance (depending on the humidity) and allow them to sit at room temperature. The agar must dry out enough to absorb the 1 ml sample students will plate.

3. Prepare the following additional materials:

   - 1-ml sterile calibrated bulbed transfer pipets (1 per student)
   - sealed tubes containing 10 to 15 ml of sterile water (1 per team)
   - alcohol wipes
   - 10 to 15 toothpicks, wrapped in aluminum foil and sterilized (1 packet per team)
   - assortment of sunscreen brands and SPF values
   - assortment of sunglasses, including those that do and do not claim protection from UV light
   - cellophane wrap in several colors (transparent, yellow, red, blue, and green)
   - fabric of varying colors and thicknesses
   - aluminum foil
   - black construction paper
   - masking or transparent tape

4. *One or two days before conducting the laboratory exercise.* Following aseptic technique, streak yeast strain G948-IC/U onto 1 YED agar plate for each team. (Again, you may want to make several extra cultures.)

   a. Hold an inoculating loop in the flame of a Bunsen burner until it is red hot.
   b. Open and flame the mouth of the yeast culture tube and touch the loop to an inside wall of the tube to cool it. Then scoop some of the yeast onto the loop. Flame the mouth of the culture tube again and replace the lid.
   c. Gently drag the loaded inoculating loop across the surface of a YED agar plate.
   d. Repeat the procedure for subsequent plates.

   Incubate the cultures in the dark overnight at 30°C.

Activity 5

Acting on Information About Cancer

At a Glance

Focus: Students assume the roles of federal legislators and explore several CD-ROM-based resources to identify reasons to support or oppose a proposed statute that would require individuals under the age of 18 to wear protective clothing when outdoors.

Major Concepts: We can use our understanding of the science of cancer to improve personal and public health. Translating our understanding of science into public policy can raise a variety of issues, such as the degree to which society should govern the health practices of individuals. Such issues often involve a tension between the values of preserving personal and public health and preserving individual freedom and autonomy.

Objectives: After completing this activity, students will

- understand that science can help us improve personal and public health,
- be able to explain that good choices can reduce an individual's risk of developing cancer and can improve an individual's chance of survival if he or she does develop it,
- understand that ethics brings to public policy debates two presumptions: that we should protect individual autonomy and that we should protect individual and societal health and well-being,
- recognize that ethical values sometimes conflict in public policy debates about strategies for reducing the risk of cancer, and
- understand that it is possible for people to hold different positions on a controversial topic and still participate in a reasoned discussion about it.

Prerequisite Knowledge: Students should understand that cancer is a disease involving uncontrolled cell division that results from mutations in genes that regulate the cell cycle. They also should understand that the genetic damage that leads to cancer accumulates across time and that exposure to agents that damage DNA can increase an individual's risk of developing cancer.

Basic Science-Public Health Connection: This activity helps students recognize that the results of scientific research can provide support for or against statutes intended to protect personal and public health.

Introduction

Approximately 1 million new cases of basal cell or squamous cell skin cancers are reported each year in the United States, and approximately 40,000 new cases of melanoma also are reported. These cancers are most common among individuals with lightly pigmented skin. Risk factors for skin cancer include excessive exposure to ultraviolet (UV) radiation, fair complexion, and occupational exposure to substances such as coal tar, creosote, arsenic compounds, and radium.

The relationship between excessive exposure to UV light and skin cancer suggests that many cases of skin cancer could be prevented by protecting skin as much as possible when outdoors. In this activity, students consider the reasons to support or oppose a proposed federal statute that would require all individuals under the age of 18 to wear headgear and clothing that covers 90 percent of their extremities while outside during peak hours of UV exposure. Discussing the relative merits of this statute offers students the opportunity to discover that one difficulty in developing public policy is that any single policy typically advances one set of interests over another. For example, enacting the statute about mandatory protective clothing advances the value of individual and societal health and well-being at the expense of the value of personal autonomy.

Materials and Preparation

You will need to prepare the following materials before conducting this activity:

- Master 5.1, *A Proposed Statute* (make 1 copy per student)
- Master 5.2, *Getting Prepared to Support or Oppose the Statute* (make 1 copy per student)
- Master 5.3, *Analyzing the Results of a Public Policy Discussion* (make 1 copy per student)
- *Cell Biology and Cancer* CD-ROM (1 per team)

Follow the instructions on pages 28–29 to load the CD-ROMs onto the computers students will use.

Note to teachers: If you do not have enough computers equipped with CD-ROM drives to conduct this activity, you can use the print-based alternative. To view and print the instructions and masters for this alternate activity, load the CD onto a computer and click the Print button on the main menu screen. The computer will display a screen showing the resources available for printing from the CD; click on the button labeled Non-CD Lesson Plan from the choices available for Activity 5, *Acting on Information About Cancer*. This will reveal the lesson plan and the masters for the alternate, non-CD-based lesson. Click Print again to print these resources.

Procedure

1. **Explain that in this activity, the students will act as elected federal legislators and members of a special committee. The committee will study the feasibility of enacting legislation to reduce the incidence of skin cancer among U.S. citizens.**

   Tip from the field test. Another way to begin the activity is to ask the students how many think they are "open-minded" and, after they have responded, to ask them what it means to be open-minded. Use probing questions to elicit the idea that being open-minded does not mean accepting all arguments or ideas as being equally valid. It *does* mean being willing to listen to and consider arguments and ideas that are different from one's own. After this discussion, introduce the activity as described in Step 1.

2. **Distribute one copy of Master 5.1, *A Proposed Statute*, to each student and ask students to organize into their teams to read and discuss the statute.**

   Initially, students may respond negatively to the statute. We recommend you not challenge this response directly, but answer with something like, "Okay, I hear your concerns. But before you decide, you should learn something about skin cancer and why this legislation has been proposed."

3. **Assign equal numbers of "pro" and "con" teams to identify reasons to support or oppose the statute. Distribute one copy of Master 5.2, *Getting Prepared to Support or Oppose the Statute*, to each student and explain that teams will have 30 minutes to study resources that will help them answer their questions about the statute and identify the key reasons to support or oppose it.**

   We recommend you assign teams to pro and con positions to assure a good balance of viewpoints during the upcoming hearing (Step 6). If students complain that they do not want to identify reasons to support a position they do not hold, explain that being able to understand and argue for positions other than their own is an important skill and will help them better understand their own position.

   Students should watch the videos on the CD-ROM (*A Proposed Statute* and *The People Respond*) and use resources in the CD-ROM-based Reference Database to help them develop their lists of reasons.

   Give the teams 30 minutes to complete their research. Reasons that students may identify include those in Figure 18. Emphasize that wherever possible, students should offer evidence in support of their reasons. For example, the statement that skin cancer is the most common type of cancer in the United States would be strengthened by citing statistics (available in the reference database) about the incidence of skin cancer.

4. **Direct the teams to identify their three strongest reasons in support of or against the statute and to designate a spokesperson to articulate those reasons.**

   Give the teams 5 minutes to complete this task.

5. **Announce that the hearing is about to begin and explain that at the end of the hearing, the class will vote on whether to recommend the statute for enactment. Emphasize that students are not required to vote for the position they were assigned to research. Instead, students should listen carefully to the discussion and decide how they will vote based on the strength of the reasons that are presented.**

6. **Begin the hearing by inviting one team that was assigned to identify reasons in support of the statute to present its position. Then, ask a team that was assigned to oppose the statute to present its position. Follow this pattern until all teams have presented their positions, then open the floor to comments and questions raised by other students.**

Science plays an important role in helping legislators make decisions about laws related to personal and public health. For example, as illustrated in this activity, science provides evidence that can be used to support or oppose laws protecting people from exposure to harmful agents. Ask students to name other examples where science has helped lawmakers act in ways that protect personal and public health (for example, mandatory vaccination programs and laws regulating toxic chemical use).

Figure 18 Reasons to Support or Oppose the Statute

To Support the Statute	To Oppose the Statute
Skin cancer is the most common type of cancer in the United States.	The statute unreasonably reduces personal freedom and may even create undue hardship.
Protection of the type described likely would reduce the incidence of UV damage that can lead to the development of skin cancer.	Although the statute applies to everyone, the risk of skin cancer is not equal for everyone.
The incidence of melanoma in the United States has more than doubled in 20 years.	It is not clear who would enforce the law or what the penalties would be.
Skin cancer carries costs for individuals and society. Potential costs include emotional costs, costs associated with the loss of productivity, insurance costs, direct costs for treatment, and costs associated with the loss of life.	It is not clear who is responsible for making sure that individuals under the age of 18 comply with the law.
As the ozone layer continues to deteriorate, the chance of experiencing harmful UV exposure increases. Although most types of skin cancer are easily detected and cured, melanoma is less easily detected in people with heavily pigmented skin and can lead to serious consequences and even death.	There are other ways to reduce the incidence of skin cancer.
	Skin cancer is easily detected and cured; the money that would be spent to enforce this statute might be better spent on widespread screening programs to detect skin cancer as early as possible.

Instruct students to continue filling in the table on *Getting Prepared* as each team presents its position. In this way, each student develops a list of reasons for and against the statute that he or she can compare prior to the class vote (Step 8).

If a team has no new reasons among its "strongest reasons" to add to the discussion, allow it to add other reasons that have not yet been presented.

7. **When it appears that students have made all the points they are prepared to make, announce that discussion on the issue is about to close. Give students 2 minutes to organize their thoughts and ask questions about any issues that they need clarified.**

8. **Designate one corner of the classroom as the area for opponents of the statute to assemble, and another corner for proponents of the statute to assemble. Ask students to vote by taking a position in the corner that reflects their position on the statute.**

   This "cornering" technique, more dramatic than voting by a show of hands, is a powerful strategy for helping students learn to take a public position on a controversial topic.

9. **Record the results of the class vote on the board.**

10. **Ask the original teams to reconvene to develop written answers to the questions on Master 5.3, *Analyzing the Results of a Public Policy Discussion.***

    Give the teams approximately 5 minutes for this task.

11. **Close the activity by inviting responses to the questions on *Analyzing the Results*.**

    Question 1 What revisions, if any, would you make to the statute in the light of the reasons you heard?

    Answers will vary. Some students may suggest that the percentage covered be reduced to make compliance less onerous and, in cases such as lifeguards, safer. Other students may suggest that certain locations, such as beaches, and certain activities, such as those that require unrestricted movement to be safe, be made exempt from the law. Still others may propose that the law apply only to people located within certain bands of latitude and/or at certain elevations.

    If students have difficulty suggesting reasonable changes, you may wish to ask them questions such as "Is there any way this law could be changed to make it acceptable to you?" or "Can the statute be modified to reduce or eliminate some of its disadvantages while keeping its important benefits?"

    Question 2 What other suggestions can you make about reducing the incidence and impact of skin cancer in the United States?

    Encourage students to think creatively here and to employ all they have learned as a result of completing the activities in this module. You may wish to point out that if they are unhappy with the proposed statute, a positive approach to defeating the measure would be to propose alternate courses of action that would have equal or greater benefits at lower cost. Students may suggest aggressive educational campaigns to alert the public, including children, to the dangers of UV exposure. They also may suggest research to develop more effective sunscreens or materials for canopies at playgrounds and beaches that let warmth and light through but block harmful UV radiation. Other possible suggestions include making annual skin cancer screening mandatory for adults over a certain age, research to develop less expensive and more effective treatment for all types of skin cancer, and even more aggressive research and policy making directed at slowing or reversing the loss of the earth's ozone layer, which is becoming an increasingly important factor in UV exposure in certain parts of the world.

    Question 3 How does this activity illustrate that

    - **good choices can reduce a person's chance of developing cancer?**

Look for evidence that students understand the importance of balancing the need for protection against the value of autonomy in personal decision making. Expect students to recognize that understanding the causes of cancer helps people make decisions about a variety of cancer-related activities, from prevention to reducing risk to detection and treatment.

Questions 3 and 4 on *Analyzing the Results* focus students' attention on the activity's major concepts.

People have many choices available to them that can significantly reduce their chances of developing skin cancer and even can increase their chances of surviving should they develop it. Some of these choices include avoiding being outdoors during hours of peak UV exposure, wearing sunscreen and protective clothing when outdoors at all, practicing regular self-examination to detect unusual changes in the skin, and seeking immediate medical care if any such changes occur.

- **values sometimes conflict in debates about laws related to personal and public health?**

  This activity illustrates the tension between trying to preserve the value of personal and public health and well-being and the value of individual autonomy.

- **it is possible for people to hold different positions on a controversial topic and still participate in a reasoned discussion about it?**

  Students should recognize that the requirement to research their assigned position, provide evidence to support their claims, and offer their ideas in a structured manner helped them discuss this issue in a rich and meaningful way. Some students may say that the discussion did not change how they voted, but most students should recognize that they have a much better understanding of the issues involved as a result of their participation.

Question 4 How has research about cancer helped improve personal and public health in the United States? Answer specifically, using examples drawn from all five of the activities in this module.

Answers will vary.

Potential Extensions

Extend or enrich this activity in the following ways.

- To help students understand how complex policy making can be, suggest that they rewrite the statute in light of the class discussion. The new statute should address the growing problem of skin cancer in a meaningful and effective way, but also should be acceptable to most students in the class.
- Invite interested students to develop, implement, and analyze the results of an informal survey that determines people's understanding or attitudes about skin cancer. Different teams of students may wish to develop quite different instruments. Be sure that students follow established practice by preserving the privacy of the survey participants.

Additional Resources for Teachers

The following resources may provide additional background information about cancer for you and your students.

Resources on the World Wide Web

National Cancer Institute (NCI)
http://rex.nci.nih.gov

This site includes sections for primary care physicians, nurse-practitioners, and other medical professionals as well as the general public.

CancerNet ™
National Cancer Institute
http://cancernet.nci.nih.gov/

This site includes sections for patients, the public, health professionals, and basic researchers. The NCI, the institute that helped support the development of this module, maintains this Web site to provide easy access to the most current information on cancer. Many of NCI's patient education resources are located here, including publications and fact sheets for cancer patients and their families.

Physician Data Query (PDQ)
http://cancernet.nci.gov/pdg.htm

This is a computerized database designed to give health professionals, patients, and the public easy access to the latest treatment, supportive care, screening, and prevention information for most types of cancer. It also provides descriptions of research studies (clinical trials) that are open for enrollment and information on organizations and physicians who specialize in cancer care. You can access PDQ through the National Library of Medicine, licensed vendors, the Information Associates Program (1–800–624–7890), or through a medical library with online searching capability. Staff at the Cancer Information Service (1–800–4–CANCER) can provide information from PDQ to callers.

American Cancer Society
http://www.cancer.org/

This is the home page for the American Cancer Society.

The Genetics of Cancer
Robert H. Lurie Comprehensive Cancer Center of Northwestern University
http://www.cancergenetics.org/

This site provides information about the genetic basis of cancer for the general public as well as primary care physicians and other medical professionals.

National Action Plan on Breast Cancer (NAPBC)
http://www.napbc.org/

The NAPBC is a public/private partnership coordinated by the Public Health Service Office on Women's Health, Department of Health and Human Services. The site includes up-to-date information about breast cancer on the World Wide Web, with links for other breast cancer-related sites.

The National Program of Cancer Registries
http://www.cdc.gov/nccdphp/dcpc/npcr/index.htm.

This site of cancer registries is maintained by the Centers for Disease Control and Prevention (CDC).

OncoLink
University of Pennsylvania Cancer Center
http://oncolink.upenn.edu/disease/breast/genetics/

This site contains a number of links to Web sites on breast cancer.

The National Alliance of Breast Cancer Organizations (NABCO)
http://www.nabco.org/

The Alliance is a coalition of organizations across the United States that offers a variety of services to breast cancer patients. It also provides information about clinical trials and breast cancer support groups.

The Prostate Cancer InfoLink
http://www.comed.com/prostate/

This site has information about prostate cancer screening, diagnosis, treatment, and support.

TeleSCAN: Telematic Services in Cancer
http://telescan.nki.nl/

This site, based in Europe, provides information to the general public, physicians, and researchers.

Books

When Life Becomes Precious by Elise Needell Babock (1997; Bantam Books; ISBN 0553378694)

This book provides information for the families and friends of cancer patients, offering advice on being supportive, explaining cancer to children, and discussing mortality. The book also includes contact information for more than 100 resources such as support organizations, hospices, and newsletters and magazines.

When a Parent Has Cancer: A Guide to Caring for Your Children by Wendy Schlessel Harpham (1997; HarperCollins; ISBN 0060187093)

This guide offers sensitive and realistic suggestions for minimizing the fear and anxiety families face when dealing with cancer.

Informed Decisions: The Complete Book of Cancer Diagnosis, Treatment, and Recovery by Gerald P. Murphy, Lois B. Morris, and Dianne Lange (1997; Viking Penguin: The American Cancer Society; ISBN 0670853704)

This rich resource explains the language of cancer, basic screening, diagnostics, and tests for cancer. It also includes information about risk factors, signs and symptoms, treatment strategies, and survival prospects. The book ends with a 170-page "Encyclopedia of Common and Uncommon Cancers" with entries that provide detailed information about a broad range of well- and little-known types of cancer.

Other Resources

Cancer Information Services (CIS)

This service is NCI's national information and education network. The CIS is an excellent source for the latest, most accurate cancer information for patients, the public, and health professionals. Specially trained staff provide scientific information in understandable language. CIS staff answer questions in English and Spanish and also distribute NCI materials. Its toll-free telephone number is 1–800–4–CANCER.

CancerMail

You can use e-mail to acquire PDQ and other NCI information by computer. To obtain a CancerMail contents list, send e-mail, with the word "help" in the body of the message, to cancermail@icicc.nci.nih.gov.

CancerFax

For NCI information by fax, dial 301–402–5874 from the telephone on a fax machine and listen to recorded instructions.

Glossary

The following glossary was modified from the glossary on the National Cancer Institute's Web site, available from http://www.nci.nih.gov.

acute lymphocytic leukemia: Type of blood cancer that originates in lymphatic cells of the bone marrow.

acute myelogenous leukemia: Type of blood cancer that involves accumulation of myeloid cells in the bone marrow and bloodstream.

adenocarcinoma: Cancer that begins in cells that line certain internal organs.

adenoma: Noncancerous tumor.

alpha-fetoprotein: Protein often found in abnormal amounts in the blood of patients with liver cancer.

Ames test: Mutagenesis assay (a measure of mutagenic ability) that involves specially engineered strains of bacteria. Because of the relationship between mutagenicity and carcinogenicity, the test is used as a rapid and relatively inexpensive first screening of untested chemicals that are suspected to be carcinogens.

anaplastic: Term used to describe cancer cells that divide rapidly and bear little or no resemblance to normal cells.

angiogenesis: Blood vessel formation, which usually accompanies the growth of malignant tissue.

angiosarcoma: Type of cancer that begins in the lining of blood vessels.

apoptosis: Normal cellular process involving a genetically programmed series of events leading to the death of a cell.

asymptomatic: Presenting no signs or symptoms of disease.

ataxia telangiectasia: Hereditary disorder characterized by problems with muscle coordination, immunodeficiency, inadequate DNA repair, and an increased risk of developing cancer.

atypical hyperplasia: Benign (noncancerous) condition in which tissue has certain abnormal features.

basal cell: Small, round cell found in the lower part, or base, of the epidermis, the outer layer of the skin.

basal cell carcinoma: Type of skin cancer that arises from the basal cells.

benign: Not cancerous; does not invade nearby tissue or spread to other parts of the body.

benign tumor: A noncancerous growth that does not spread to other parts of the body.

biological therapy: Use of the body's immune system, either directly or indirectly, to fight cancer or to lessen side effects that may be caused by some cancer treatments. Also known as immuno-therapy, biotherapy, or biological response modifier therapy.

biopsy: Removal of a sample of tissue, which is then examined under a microscope to check for cancer cells.

bone marrow: Soft, spongy tissue in the center of large bones that produces white blood cells, red blood cells, and platelets.

bone marrow aspiration: Removal of a small sample of bone marrow (usually from the hip) through a needle for examination under a microscope to see whether cancer cells are present.

bone marrow biopsy: Removal of a sample of tissue from the bone marrow with a large needle. The cells are checked to see whether they are cancerous. If cancerous plasma cells are found, the pathologist estimates how much of the bone marrow is affected. Bone marrow biopsy is usually done at the same time as bone marrow aspiration.

bone marrow transplantation: Procedure in which doctors replace marrow destroyed by treatment with high doses of anticancer drugs or radiation. The replacement marrow may be taken from the patient before treatment or may be donated by another person.

bone scan: Technique to create images of bones on a computer screen or on film. A small amount of radioactive material is injected and travels through the bloodstream. It collects in the bones, especially in abnormal areas of the bones, and is detected by a scanner.

brachytherapy: Internal radiation therapy using an implant of radioactive material placed directly into or near the tumor.

***BRCA1*:** Gene located on chromosome 17 that normally helps restrain cell growth. Inheriting an altered version of *BRCA1* predisposes an individual to breast, ovarian, or prostate cancer.

***BRCA2*:** Gene located on chromosome 13 that scientists believe may account for 30 to 40 percent of all inherited breast cancer.

breast reconstruction: Surgery to rebuild a breast's shape after a mastectomy.

Burkitt lymphoma: Type of non-Hodgkin lymphoma that most often occurs in young people between the ages of 12 and 30. The disease usually causes a rapidly growing tumor in the abdomen.

cancer: Term for a group of more than 100 diseases in which abnormal cells divide without control. Cancer cells can invade nearby tissues and can spread through the bloodstream and lymphocytic system to other parts of the body.

carcinogen: Any substance that is known to cause cancer.

carcinogenesis: Process by which normal cells are transformed into cancer cells.

carcinoma: Cancer that begins in the lining or covering of an organ.

carcinoma in situ: Cancer that involves only the cells in which it began and has not spread to other tissues.

CEA assay: Laboratory test to measure the level of carcinoembryonic antigen (CEA), a substance that is sometimes found in an increased amount in the blood of patients with certain cancers.

cell cycle: Sequence of events by which cells enlarge and divide. Includes stages typically named G1, S, G2, and M.

chemoprevention: Use of natural or laboratory-made substances to prevent cancer.

chemotherapy: Treatment with anticancer drugs.

chronic lymphocytic leukemia: Type of blood cancer that involves overproduction of mature lymphocytes.

chronic myelogenous leukemia: Type of blood cancer that involves accumulation of granulocytes (a type of white blood cell) in the bone marrow and bloodstream.

clinical trial: Research study that involves patients. Each study is designed to find better ways to prevent, detect, diagnose, or treat cancer and to answer scientific questions.

colonoscopy: Procedure that uses a flexible fiber optic endoscope to examine the internal surface of the colon along its entire length.

combination chemotherapy: Treatment in which two or more chemicals are used to obtain more effective results.

computed tomography: X-ray procedure that uses a computer to produce a detailed picture of a cross section of the body; also called CAT or CT scan.

contact inhibition: Inhibition of cell division in normal (noncancerous) cells when they contact a neighboring cell.

CT (or CAT) scan: *See* computed tomography.

cytotoxic: Poisonous to cells. In chemotherapy, used to describe an agent that is poisonous to cancer cells.

diagnosis: Process of identifying a disease by the signs and symptoms.

dysplasia: Abnormal cells that are not cancer.

dysplastic nevi: Atypical moles; moles whose appearance is different from that of common moles. Dysplastic nevi are generally larger than ordinary moles and have irregular and indistinct borders. Their color often is not uniform and ranges from pink or even white to dark brown or black; they usually are flat, but parts may be raised above the skin surface.

encapsulated: Confined to a specific area; an encapsulated tumor remains in a compact form.

endometrial: Having to do with the mucous membrane that lines the cavity of the uterus.

environmental tobacco smoke: Smoke that comes from the burning end of a cigarette and smoke that is exhaled by smokers. Also called ETS or secondhand smoke. Inhaling ETS is called involuntary or passive smoking.

epidemiology: Study of the factors that affect the prevalence, distribution, and control of disease.

epidermis: Upper or outer layer of the two main layers of cells that make up the skin.

Epstein-Barr virus: Virus that has been associated with the development of infectious mononucleosis and also with Burkitt lymphoma.

estrogen: Female hormone produced by the ovary. Responsible for secondary sex characteristics and cyclic changes in the lining of the uterus and vagina.

etiology: Study of the causes of abnormal condition or disease.

familial polyposis: Inherited condition in which several hundred polyps develop in the colon and rectum. These polyps have a high potential to become malignant.

fecal occult blood test: Test to reveal blood hidden in the feces, which may be a sign of colon cancer.

fiber: Parts of fruits and vegetables that cannot be digested. Also called bulk or roughage.

fibroid: Benign uterine tumor made up of fibrous and muscular tissue.

gene therapy: Treatment that alters genes (the basic units of heredity found in all cells in the body). In studies of gene therapy for cancer, researchers are trying to improve the body's natural ability to fight the disease or to make the tumor more sensitive to other kinds of therapy.

genetic: Inherited; having to do with information that is passed from parents to children through DNA in the genes.

grade: Describes how closely a cancer resembles normal tissue of its same type, along with the cancer's probable rate of growth.

grading: System for classifying cancer cells in terms of how malignant or aggressive they appear microscopically. The grading of a tumor indicates how quickly cancer cells are likely to spread and plays a role in treatment decisions.

herpes virus: Member of the herpes family of viruses. One type of herpes virus is sexually transmitted and causes sores on the genitals.

hormonal therapy: Treatment of cancer by removing, blocking, or adding hormones.

human papillomaviruses: Viruses that generally cause warts. Some papillomaviruses are sexually transmitted. Some of these sexually transmitted viruses cause wartlike growths on the genitals, and some are thought to cause abnormal changes in cells of the cervix.

hyperplasia: Precancerous condition in which there is an increase in the number of normal cells lining an organ.

imaging: Tests that produce pictures of areas inside the body.

immunotherapy: Treatment that uses the body's natural defenses to fight cancer. Also called biotherapy or biological modifier response therapy.

incidence: Number of new cases of a disease diagnosed each year.

incidence rate: Number of new cases per year per 100,000 persons.

initiation: Preneoplastic change in the genetic material of cells caused by a chemical carcinogen. Cancer develops when initiated cells are subsequently exposed to the same or another carcinogen.

in situ cancer: Cancer that has remained within the tissue in which it originated.

invasion: As related to cancer, the spread of cancer cells into healthy tissue adjacent to the tumor.

invasive cancer: Cancer that has spread beyond the layer of tissue in which it developed.

keratin: Insoluble protein that is the major constituent of the outer layer of the skin, nails, and hair.

lesion: Area of abnormal tissue change.

leukemia: Cancer of the blood cells.

lifetime risk: Probability that a person, over the course of a lifetime, will develop cancer.

Li-Fraumeni syndrome: Rare family predisposition to multiple cancers, caused by an alteration in the *p53* tumor suppressor gene.

lumen: An enclosed space bounded by an epithelial membrane; for example, the lumen of the gut.

malignant: Cancerous; can invade nearby tissue and spread to other parts of the body.

melanin: Skin pigment (substance that gives the skin its color). Dark-skinned people have more melanin than light-skinned people.

melanocyte: Cell in the skin that produces and contains the pigment called melanin.

melanoma: Cancer of the cells that produce pigment in the skin. Melanoma usually begins in a mole.

metastasis: Cancer growth (secondary tumors) that is anatomically separated from the site at which the original cancer developed.

metastasize: To spread from one part of the body to another. When cancer cells metastasize and form secondary tumors, the cells in the metastatic tumor are like those in the original (primary) tumor.

mole: Area on the skin (usually dark in color) that contains a cluster of melanocytes. *See also* nevus.

monoclonal: Population of cells that was derived by cell division from a single ancestral cell.

morbidity: Disease.

mortality: Death.

mortality rate: Number of deaths per 100,000 persons per year.

mutagen: Any substance that is known to cause mutations.

mutagenesis: Process by which mutations occur.

mutation: Change in the way cells function or develop, caused by an inherited genetic defect or an environmental exposure. Such changes may lead to cancer.

National Cancer Institute (NCI): The largest of the 24 separate institutes, centers, and divisions of the National Institutes of Health. The NCI coordinates the federal government's cancer research program.

National Institutes of Health (NIH): One of eight health agencies of the Public Health Service (the Public Health Service is part of the U.S. Department of Health and Human Services). Composed of 24 separate institutes, centers, and divisions, NIH is the largest biomedical research facility in the world.

necrosis: Cell death.

neoplasia: Abnormal new growth of cells.

neoplasm: New growth of tissue. Can be referred to as benign or malignant.

nevus: Medical term for a spot on the skin, such as a mole. A mole is a cluster of melanocytes that usually appears as a dark spot on the skin.

non-Hodgkin lymphoma: One of the several types of lymphoma (cancer that develops in the lymphocytic system) that are not Hodgkin lymphoma. Hodgkin lymphoma is rare and occurs most often in people aged 15 to 34 and in people over 55. All

other lymphomas are grouped together and called non-Hodgkin lymphoma.

nonmelanoma skin cancer: Skin cancer that does not involve melanocytes. Basal cell cancer and squamous cell cancer are nonmelanoma skin cancers.

Office of Science Education (OSE): The Office of Science Education of the National Institutes of Health (NIH) coordinates science education activities at NIH and sponsors science education projects in-house.

oncogene: Gene that normally directs cell growth but also can promote or allow the uncontrolled growth of cancer if damaged (mutated) by an environmental exposure to carcinogens or if damaged or missing because of an inherited defect.

oncogenic: Having the capacity to cause cancer.

oncologist: Doctor who specializes in treating cancer. Some oncologists specialize in a particular type of cancer treatment. For example, a radiation oncologist specializes in treating cancer with radiation.

oncology: Study of tumors encompassing their physical, chemical, and biologic properties.

oopherectomy: Surgical removal of one or both ovaries.

p53: Gene that normally inhibits the growth of tumors, which can prevent or slow the spread of cancer.

palliative treatment: Treatment that does not alter the course of a disease, but improves the quality of life.

polyclonal: Population of cells that was derived by cell division from more than one ancestral cell.

polyp: Mass of tissue that projects into the colon.

precancerous: Term used to describe a condition that may or is likely to become cancer.

precancerous polyp: Growths in the colon that often become cancerous.

progesterone: Female hormone produced by the ovaries and placenta; responsible for preparing the uterine lining for implantation of an early embryo.

prognosis: Probable outcome or course of a disease; the chance of recovery.

promotion: Expression of the cancerous potential of initiated cells after exposure to the same or a different carcinogen.

prophylactic: Treatment administered or taken to prevent disease.

proto-oncogene: Gene that, when converted to an oncogene by a mutation or other change, can cause a normal cell to become malignant. Normal oncogenes function to control normal cell growth and differentiation.

radiation therapy: Treatment with high-energy rays (such as X-rays) to kill cancer cells. The radiation may come from outside the body (external radiation) or from radioactive materials placed directly in the tumor (implant radiation). Also called radiotherapy.

radioactive: Giving off radiation.

radon: Radioactive gas that is released by uranium, a substance found in soil and rock. When too much radon is breathed in, it can damage lung cells and lead to lung cancer.

relative risk: Comparison of the risk of developing cancer in persons with a certain type of exposure or characteristic with the risk in persons who do not have this exposure or characteristic.

remission: Disappearance of the signs and symptoms of cancer. When this happens, the disease is said to be "in remission." A remission can be temporary or permanent.

retinoblastoma: Eye cancer caused by the loss of both copies of the tumor suppressor gene *RB;* the inherited form typically occurs in childhood because one gene is missing from the time of birth.

retrovirus: Small RNA virus that has an RNA genome. Acts as a template for the production of the DNA that is integrated into the DNA of the host cell. Many retroviruses are believed to be oncogenic.

risk factor: Something that increases the chance of developing a disease.

Rous sarcoma virus: Chicken retrovirus that was the first virus shown to cause a malignancy.

sarcoma: Malignant tumor that begins in connective and supportive tissue.

screening: Checking for disease when there are no symptoms.

secondary tumor: Metastasis.

SEER Program: Surveillance, Epidemiology, and End Results Program of the National Cancer Institute. Started in 1973, SEER collects cancer incidence data in eleven geographic areas and two supplemental registries, for a combined population of approximately 14 percent of the total U.S. population.

side effect: Problem that occurs when treatment affects healthy cells. Common side effects of cancer treatment are fatigue, nausea, vomiting, decreased blood cell counts, hair loss, and mouth sores.

somatic cell: Any of the body cells except the reproductive cells.

SPF (sun protection factor): Scale for rating sunscreens. Sunscreens with an SPF of 15 or higher provide the best protection from the sun's harmful rays.

squamous cell cancer: Type of skin cancer that arises from the squamous cells.

stage: Extent of a cancer, especially whether the disease has spread from the original site to other parts of the body.

staging: Doing exams and tests to learn the extent of the cancer, especially whether it has spread from its original site to other parts of the body.

stem cells: Cells from which all blood cells develop.

sun protection factor: *See* SPF.

sunscreen: Substance that blocks the effect of the sun's harmful rays. Using lotions or creams that contain sunscreens can protect the skin from damage that may lead to cancer. *See also* SPF.

survival rate: Proportion of patients alive at some point after their diagnosis of a disease.

telomerase: Enzyme that is present and active in cells that can divide without apparent limit (for example, cancer cells and cells of the germ line). Telomerase replaces the missing repeated sequences of each telomere.

telomere: End of a chromosome. In vertebrate cells, each telomere consists of thousands of copies of the same DNA sequence, repeated again and again. Telomeres become shorter each time a cell divides; when one or more telomeres reaches a minimum length, cell division stops. This mechanism limits the number of times a cell can divide.

testosterone: Male sex hormone.

transformation: Change that a normal cell undergoes as it becomes malignant.

tumor: Abnormal mass of tissue that results from excessive cell division. Tumors perform no useful body function. They may be either benign (not cancerous) or malignant (cancerous).

tumor marker: Substance in blood or other body fluids that may suggest that a person has cancer.

tumor suppressor gene: Gene in the body that can suppress or block the development of cancer.

ultraviolet (UV) radiation: Invisible rays that are part of the energy that comes from the sun. UV radiation can burn the skin and cause melanoma and other types of skin cancer. UV radiation that reaches the earth's surface is made up of two types of rays, UVA and UVB rays. UVB rays are more likely than UVA rays to cause sunburn, but UVA rays pass further into the skin. Scientists have long thought that UVB radiation can cause melanoma and other types of skin cancer. They now think that UVA radiation also may add to skin damage that can lead to cancer. For this reason, skin specialists recommend that people use sunscreens that block or absorb both kinds of UV radiation.

X-chromosome inactivation: Process by which one of the two X chromosomes in each cell from a female mammal becomes condensed and inactive.

This process assures that most genes on the X chromosome are expressed to the same extent in both males and females.

X-ray: High-energy radiation used in low doses to diagnose diseases and in high doses to treat cancer.

xeroderma pigmentosum: Hereditary disease characterized by extreme sensitivity to the sun and a tendency to develop skin cancers. Caused by inadequate DNA repair.

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Masters

Activity 1, *The Faces of Cancer*

Master 1.1, *The Faces of Cancer* ..... classroom set
Master 1.2, *Team Summary* ..... student copies
Master 1.3, *Drawing Conclusions from the Faces of Cancer* ..... student copies
Master 1.4, *Summary Profile of the Faces of Cancer* ..... transparency

Activity 2, *Cancer and the Cell Cycle*

Master 2.1, *Understanding Cancer* ..... student copies

Activity 3, *Cancer as a Multistep Process*

Master 3.1, *Colon Cancer Incidence by Age* ..... student copies and transparency
Master 3.2, *Random Number Tables* ..... classroom set
Master 3.3, *Collecting the Data* ..... transparency
Master 3.4, *Graphing the Data* ..... student copies and transparency
Master 3.5, *Using the Hit Simulator* ..... student copies
Master 3.6, *Testing an Explanation by Looking at Additional Data* ..... student copies

Activity 4, *Evaluating Claims About Cancer*

Master 4.1, *Media Item 1* ..... student copies
Master 4.2, *Media Item 2* ..... student copies
Master 4.3, *Media Item 3* ..... student copies
Master 4.4, *Media Item 4* ..... student copies
Master 4.5, *Using a Model System to Test Claims About UV Light* ..... student copies
Master 4.6, *Evaluating Claims About Cancer* ..... student copies

Activity 5, *Acting on Information About Cancer*

Master 5.1, *A Proposed Statute* ..... student copies
Master 5.2, *Getting Prepared to Support or Oppose the Statute* ..... student copies
Master 5.3, *Analyzing the Results of a Public Policy Discussion* ..... student copies

The Faces of Cancer

Si Conners
I was born in 1925 in Texas of African-American parents. My mother had breast cancer; otherwise there was no history of cancer in my family.

0–19 years

I was a willing, anxious student, hard working and eager to please. I was the last of four children and, early on, developed the skills of getting along with others and negotiating for what I want. I was 12 when my mother was diagnosed with breast cancer, and I helped with her care when she came home from the hospital. My older sister also had sickle cell disease. Because I helped take care of her, I was familiar with the health care system in my community and understood how to obtain medical advice when I needed it.

20–39 years

I went to the university in Houston to earn my bachelor's degree in history, then went on to study law. After I passed the bar, I worked as an advocate for the NAACP. My wife and I had no children, but I was active in the church youth group and coached Little League.

40–59 years

My first serious health problem was diagnosed as diabetes at age 48. Initially, it was hard to control, and I saw an internist on a regular basis. Even after the diabetes was controlled with insulin, there was some concern about my kidney function, so I continued regular check-ups.

60+ years

Despite my regular check-ups for diabetes and high blood pressure (I had developed this by age 65), I had not undergone cancer screening and was beginning to think I should. When the community offered free screening, I took advantage of the opportunity and, at age 70, was diagnosed with prostate cancer. Prostate cancer is believed to grow slowly, and because of my age, I did not want to undergo surgery or radiation, so I chose to be treated only with hormones.

Si died at age 73 from diabetes. Many of the families whose children he had coached in Little League came to his funeral.

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Rosemarie Winters
I was born in 1932 in West Virginia of wealthy Caucasian parents. My family had a history of high blood pressure and heart disease, but not of cancer.

0–19 years

I was only an average student in school, although my teachers consistently told my parents I could do better if I tried. Both my parents smoked. Not surprisingly perhaps, I began smoking as well at age 16. I was plagued with allergies and mild asthma through school and was never interested in sports. Instead, I loved to socialize, and even as a teenager, I was a moderate beer drinker.

20–39 years

I joined the work force instead of going to college or getting married right out of high school. As a secretary, I worked hard during the week and partied hard on the weekends. I still smoked, despite several attempts to stop.

I was married at 27 and had a child at 29. I never remarried after my divorce at age 36.

40–59 years

By 45, I had developed significant bronchial problems. I had a persistent cough and frequent bouts with deep chest colds and congestion. I finally stopped smoking at age 55, but my hacking cough persisted.

60+ years

I was diagnosed with lung cancer at age 63. By the time the cancer was detected, it had metastasized to my brain and was producing headaches, nausea, and vomiting. My doctor gave me chemotherapy and radiation, which made me feel tired and sick, but the cancer never went away.

Rosemarie died three months later just a little shy of her 64th birthday. Her family grieved, but was not surprised. Rosemarie's doctor had told them that the five-year survival rate for lung cancer is only about 13 percent.

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Brian Eaken
I was born in 1935 in Chicago to middle-class, African-American parents. My family had no history of cancer, though my father smoked till he died of a stroke at age 70.

0–19 years

I was an inconsistent student, excelling in subjects I enjoyed and ignoring subjects that didn't interest me. I didn't smoke or drink as a teenager, and, aside from periodic allergies (allergies possibly secondary to my father's smoking), I enjoyed generally good health. I loved sports, especially baseball, and won a scholarship to play ball for a local college.

20–39 years

I was injured during my third year of college baseball and eventually stopped playing. I began to drink and smoke and socialize, was married soon after finishing college, and divorced a few years later. After my divorce, I moved into a small apartment and focused most of my energies on work and success. I knew that I ate unhealthily but really didn't have time to fuss with meals.

40–59 years

By age 40, I was on medication for high blood pressure; by age 46, I was taking insulin to control my diabetes. Eventually, I developed a cough that wouldn't go away and finally was diagnosed with emphysema. My poor health forced me to slow down, and by my late 50s, I was semiretired.

60+ years

My stroke at age 60 forced my full retirement. An X-ray a year later showed small spots in one lung, which were surgically removed.

Brian seemed to be recovering well from the lung cancer, but died at age 63 from a second, serious stroke.

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Mario Devencenzi
I was born in 1932 in northern Minnesota, of Italian ancestry. I was the second oldest of eight children in a relatively poor family, so I felt some real responsibility to help my family. My family had no history of cancer.

0–19 years

I was an average student in school, but in response to tough financial times in my family, I dropped out of school at age 10 to take a job in the ore mines. I felt good to be able to bring my pay home each week and to be able to hold my own in the rough-and-tumble environment of the mines. By age 13, I had started smoking like many of the men I worked with; by age 15, I was up to three packs a day.

20–39 years

I continued to work in the mines after I was married. Although my first marriage ended in divorce a few years later, I married again and eventually had six children, one of whom died at age 2 of an infection. I was generally too busy to worry about my health. Besides, most of the time, I felt just fine! My primary interests in life were cards, TV, and drinking with the guys.

40–59 years

By my mid-40s, I had developed a persistent cough and was starting to feel chronically fatigued or in my words, "to feel my age." Welcome relief from my normally strenuous work came at age 48, when I was promoted to supervisor. Now I wasn't in the mines as much, but instead spent most of my time behind a desk.

At age 59, I noticed with alarm that there was blood in the stuff I was coughing up out of my lungs. Worried now, I went to the company doctor, who ordered X-rays. X-rays revealed cancer that involved significant portions of both of my lungs and was too advanced for surgery. Eventually, I was too sick to work.

60+ years

Although I was too sick to work, I did go to chemotherapy and radiation treatments. My doctor also tried surgery, but I continued to feel worse.

Mario died at age 61. He was survived by 5 children and 12 grandchildren.

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Kathy Becker
I was born in Kansas in 1976 of Caucasian parents. My family was middle class, with four children (I was the youngest). There was no history of cancer in my family.

0–19 years

My childhood was unremarkable. I was a good student, had no major childhood illnesses, but had the flu about every other year. I never smoked or drank. I participated in sports, but was often tired.

When I was 14, I dropped out of sports because I was so tired. I also bruised easily and sometimes experienced pain in my bones. My mother noticed my bruises, and two months later, my parents took me to a pediatrician in response to anorexia and abdominal pain. My physical examination and blood cell counts indicated leukemia; I was referred to University Hospital for appropriate therapy. I was lucky I got involved in a clinical trial and got the very best of care! It paid off, too, with remission and a good chance for a healthy, productive life even after cancer.

20–39 years

At age 20, I was still in remission (cancer-free, and it had been six years since my treatment). After I finished college, I joined the Peace Corps and traveled to South America where I taught school and helped a rural community establish a small library and a computer center. I returned home at age 28 and settled down in Kansas to lead a "quieter" life.

40–59 years

I was happy as a school teacher and part-time librarian and never missed being married. I traveled each year—sometimes to Europe, other times to Asia or South America—and continued supporting charitable organizations that built schools and libraries for poor children around the world.

60+ years

I retired at age 60 and returned to visit the little town in South America where I served in the Peace Corps so many years ago. What a change the 21st century has brought to those people. Connected now to the rest of the world by the Web, you would think they'd have little need for the pitifully small library I helped them build. Still, they cherish the books (especially the children's books), and I feel good that my efforts are still yielding benefits for the people I knew when I was there, and for their children.

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Cherisse Nicholson
I was born in 1950 in Arizona, of European ancestry. My family was well off financially, and I was always encouraged to set my sights high and live life to the fullest. My father died of prostate cancer at age 74, and my mother's sister died of breast cancer.

0–19 years

I had a carefree childhood, growing up in sunny Arizona. Despite my fair skin and freckles, I loved the sun and spent lots of time outdoors. My father loved golf, and I usually caddied for him. I also spent my summers in California on the beach. After I graduated from high school, I headed off to college to major in political science.

20–39 years

My good health continued through college and after. Although I started smoking in college, I watched what I ate, got lots of exercise, and kept my smoking to four or five cigarettes per day. I took birth control pills from age 17 to after I was married; then, after my marriage and career as a congressional aide were established, I had three children (at ages 26, 29, and 34). My one serious sorrow was my three miscarriages (at ages 31, 32, and 36). My husband and I love children and would have been happy to have had 10!

40–59 years

Despite my family, I continued an active professional life. My golfing background came in handy as I was the one golfing with the business people and politicians I needed to network with. I continued to take care of myself and got regular check-ups..

In my late 40s, I noticed dark "liver spots" on my hands, shoulders, and nose. I also began to worry about what I perceived to be excessive wrinkling, probably from years of being in the sun. My doctor noticed the spots too, but I was worried about scarring and didn't want them removed. By age 54, some of the spots were getting larger, and when I was 56, I had them removed. Though the diagnosis was definitely skin cancer, the doctor told me the margins were clear and the outlook was good.

60+ years

I am retired now, but continue an active life. Since my initial bout with skin cancer, I have had 11 more discolored spots on my skin removed, but have had no other health problems. I faithfully visit the doctor for check-ups twice a year and now wear sunscreen when I am outside, even when the day is cloudy!

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Carlos Montano
I was born in 1937 in Southern California of Mexican immigrants. My father's aunt and my mother both had cancer, but I am not sure what type.

0–19 years

As the son of immigrant migrant workers, I spent lots of time in the fields. I attended school only sporadically and almost never went to the doctor. As a consequence, I received only some of my childhood immunizations and got used to the habit of going to the emergency room only when I had serious problems.

Life was tough during this time, but my family was close-knit and we survived.

20–39 years

As an adult, I finally settled down in one place, got married, and started to raise a family. I finished my GED and continued working in agriculture, eventually becoming a supervisor on a large commercial farm. My three children attended school regularly, and I became involved in a variety of community initiatives, including one designed to reach all local children with information about the importance of washing one's hands and with the opportunity to receive proper immunizations.

40–59 years

I remained active in the community, especially in programs that improved access to health information (such as, information about pesticide use and sun exposure) and gave farm workers and their children access to health care. At age 59, I participated in a community health cancer screening and had three moles removed from my back. They proved to be noncancerous, but the screening also revealed early prostate cancer, which my doctor immediately treated with surgery and radiation therapy.

60+ years

I am definitely feeling my age these days, but I have a positive outlook on life. My doctor says I am doing "OK," and I interpret that to mean I am on my way to full recovery.

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Maria Delgano
I was born in 1939 in Washington, DC, of Puerto Rican parents. My family had no history of cancer, though my father had smoked for his entire adult life.

0–19 years

My family was poor, and I grew up angry and rebellious at what I perceived to be life's injustices. I was a poor student in school, and by 14 was smoking two to three packs of cigarettes a day. By 15, I was drinking with my friends; at age 17, I was hospitalized a couple of times for injuries related to street fights.

20–39 years

As an adult, I finally found a job as a transport clerk in the subway system. The job paid reasonably well and came with a good health care plan, but I was busy with my four children and did not regularly seek health advice. I knew the doctor would tell me to eat better and to stop smoking and, for sure, I didn't want to hear that!

I developed a troublesome cough in my mid-30s, but explained it away as the result of the damp environment of the subway system. By then, I was wishing I had a different type of job, but I didn't really have any skills or qualifications. Because I couldn't afford to quit working, I stuck it out.

40–59 years

As I aged, my coughing and wheezing continued to be a problem, especially during the three to four times a year when I had a cold or the flu and couldn't breathe. I stayed active at work and in my family, until age 59, when a serious attack of chest pain sent me to the emergency room. Subsequent X-rays revealed lung cancer, with metastasis to my bones.

60+ years

Doctors treated my cancer with chemotherapy, not so much as a cure, but to improve my survival time. They warned, though, that even if the cancer responded well to the treatment, it likely would return within a few years.

Today, I live with my oldest daughter, who scolds me when I do too much and trigger an attack of coughing and chest pain. I am proud of my family's accomplishments and am hoping to be present when my oldest grandson graduates from college next year.

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Ed Manning
I was born in 1930 in Kentucky, son of Caucasian parents Katie and Jim Manning. My mother died of breast cancer at age 47, and my father died from a stroke at age 61.

0–19 years

I had a normal childhood in rural Kentucky. I was an average student despite frequent absences due to flulike symptoms and fevers. My doctor told my parents that my immune system may not be very robust, but my mother ascribed my frequent illnesses to my distaste for vegetables. Fruit was okay, but vegetables? Never!

20–39 years

As I got older, I continued to suffer with colds and flu, often having as many as five to six bouts with it a year. I never smoked. I always thought it was bad business for a salesman to smoke and I drank minimally. My wife and I had two children and loved to fish and sail with them all summer.

40–59 years

As I aged, I began having other health problems. At age 42, I was diagnosed with diabetes and put on a strict diet. My doctor also noted my chronically high blood pressure and prescribed medication to control it. By my late 40s and early 50s, I had also developed several allergies.

Despite my health problems, these were good years as I watched my children grow up, get married, and start families of their own.

60+ years

I was diagnosed with skin cancer at age 62; after my doctor removed the two or three discolored moles on my arms, I had no further problems. In fact, today I feel as good as I ever did. The changes that I *have* introduced into my life are that now I eat my vegetables and I also wear sunscreen!

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Margaret Alexander
I was born in 1957 in Utah, of European ancestry. Several members of my family have had cancer: My older sister had breast cancer at age 36, my mother had ovarian cancer at age 49, my mother's sister had breast cancer at age 47, and my mother's mother had breast cancer at age 54.

0–19 years

My childhood was uneventful. I met my future husband my first semester in college and was married at age 19. A few months after my wedding, I was pregnant.

20–39 years

My first child was born when I was just 20, and two more followed at ages 24 and 27. I breastfed each baby for about six months.

Despite the children, I finished my nursing degree and got a job as a nurse in a local hospital. Aware of the history of cancer in my family, I regularly practiced breast self-examination. I was not referred for a mammogram because I was considered too young.

At age 32, I agreed to participate in a research study. In 1996, as part of that study, I learned that I carry an altered gene that increases my risk for breast and ovarian cancer. On the strength of this new information, I was able to get mammograms at regular intervals.

40–59 years

At age 42, my mammogram showed some suspicious calcifications. A biopsy revealed breast cancer. I had a partial mastectomy with 12 lymph nodes removed. Because 3 of the 12 nodes showed cancer, I had both radiation and chemotherapy and was put on Tamoxifen (a drug that reduces the chances of breast cancer developing) for five years.

60+ years

I feel great these days and am grateful that the research study led to my cancer being detected early. I continue my practice of regular self-examination and see my doctor twice a year. I have had no recurrences of the breast cancer and feel confident of the future. Because the altered gene I carry also predisposes me to increased risk of ovarian cancer, I considered having my ovaries removed, but finally decided not to take that step. But, I will have regular check-ups!

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Leila Johnson
I was born in 1929 in Los Angeles of African-American parents. My father was part owner of a small corner grocery store, and my mother taught piano lessons. My family had no history of cancer.

0–19 years

I was a quiet child who loved to read and to write poetry and short plays, which my friends and I would stage. I was a good student and neither smoked nor drank. After finishing high school, I started college, majoring in English literature.

20–39 years

I finished my degree and, at age 22, married my childhood sweetheart. My husband and I moved to rural Georgia, where I taught high school during the school year and gardened during the summer. Because of regular insect invasions, I used pesticides often, but always washed my vegetables before eating them. I also oversaw the pesticide spraying that I sometimes hired people to do in my gardens.

After two miscarriages, I had my first child at age 28.

40–59 years

I had my second and third children at ages 31 and 37. Although I breastfed the first two children for a year each, I didn't breastfeed the third.

Because of the distance I had to travel to see my doctor, I had only sporadic health care most of my life. I examined my breasts when I remembered to—perhaps two or three times a year—but typically I did not have regular clinical examinations.

Mammograms at ages 50 and 56 were normal; I experienced menopause at age 53 and went on hormone replacement therapy.

60+ years

I found my first lump in my breast when I was 63. When I saw the doctor six months later I was diagnosed with breast cancer. By then, the treatment was full mastectomy. After my surgery, I was put on a schedule of radiation therapy and chemotherapy, then started on Tamoxifen, a drug that reduces the risk of developing breast cancer. Today, I am doing poorly—the cancer has metastasized to my liver and is not responding to therapy.

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Avi Rothstein
I was born in 1950 in New York City. My family was of Eastern German descent; sadly, most of them died in the Holocaust. One uncle, who also lives in New York, seems hale and hearty.

0–19 years

I grew up in a poor neighborhood and didn't get regular health care. Despite this, I was a healthy child (though my mother often scolded me for not eating my vegetables). I was raised on a traditional Kosher diet; I didn't smoke, even as a teenager, and drank only occasionally.

20–39 years

After college, I went to law school and then lived and worked as a lawyer in New York. My job carried very high stress, and I had little time to relax. I even ate on the run, except for rare weekends that I spent on Long Island with my wife's family.

40–59 years

At age 41, I saw a doctor about my stomach. I had long suffered from an irritable bowel, but when the doctor found blood in my stool, even I was concerned. A subsequent colonoscopy revealed several polyps in my colon—one cancerous.

The cancer had not spread, so the surgeon was able to remove it completely. The experience prompted me to have colonscopies every other year. I want to catch the polyps before they can become cancerous.

When my uncle heard I had colon cancer, he decided to go in for colon screening. When he was found to have polyps too, we both enrolled in a genetic testing program to see whether we carried predisposing genes. Both of us have a predisposing mutation, one of several known to increase risk of colon cancer.

60+ years

I am doing well these days, but now I eat my vegetables and see my doctor regularly.

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Elizabeth Gries
I was born in Milwaukee, the youngest of three children of a Polish mother and a German father. My father worked as a supervisor in a factory, and we lived in a nice but not fancy part of town. My father's sister died of lung cancer when she was in her 60s (she was a heavy smoker); my father died of a heart attack in his late 40s.

0–19 years

I was an average student in school and considered myself pretty healthy and happy. I always ate well (I loved fried foods and meat), got lots of sleep, and had regular check-ups. In fact, my only serious health problem as a child was a broken leg when I was 11. (My grandparents never forgave me for jumping down from the loft in the barn on their farm . . .)

20–39 years

I got a job as a secretary for a car parts manufacturer after high school, but quit after I was married at age 23. I drank a little (just socially) and also smoked just a little (maybe a pack a day). I had three children (at ages 26, 29, and 33) who kept me busy and active. I was always very healthy (though I tended to be a little overweight) and saw my doctor at least once a year for a check-up.

40–59 years

I developed high blood pressure and high cholesterol in my early 40s, which I struggled to control by changing my diet. When I was 45, my doctor finally put me on medication to control these problems. But when I was 53, I had a heart attack. You can believe I stopped smoking then fast! I also tried even harder to eat a lower-fat diet. After menopause (I was 56), my doctor put me on hormone replacement therapy, but it made me gain weight and eventually he said I could stop taking it (I was 59).

60+ years

After my husband died, I sold my home, moved into an apartment, and spent lots of time with my children and grandchildren. I continued seeing the doctor regularly and, when I was 79, a routine mammogram revealed breast cancer. Luckily (especially since I didn't examine my breasts very often), we caught it early. I had a lumpectomy and radiation therapy and had no problems from it afterward.

Elizabeth died at age 83 from a second heart attack. She was survived by three children and seven grandchildren.

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Mark Harris
I was an African-American man born of middle-class parents who lived in Detroit, Michigan. My only sister died of lung cancer when she was in her 50s, but I don't know of any cancer in the family except that.

0–19 years

I didn't like school much, though I was a good student when I tried. I gave my mom some problems sometimes (mostly I just got in trouble for skipping school or fighting) and quit school when I was 16 to work on a production line for an automobile manufacturer. I didn't take care of myself those days—I partied a lot on the weekends and often forgot to eat.

20–39 years

The company I worked for encouraged me to get my GED and, in my early 30s, I was promoted to a supervisory position. Life was good. I was busy with a home and family (a wife and four kids), but found time now and then to do some woodworking and metal sculpture as a hobby. The company provided good benefits, including a company-provided physical each year, so I stayed pretty healthy. The doctor discovered I had high blood pressure, but the medicine I took controlled it and I didn't worry.

40–59 years

My interest in woodworking and sculpture really took off after my children left home and I retired (age 55). I won a couple of amateur art awards and also developed an interest in traveling.

After a few years of suffering through the Michigan winters, my wife and I moved to Arizona, where I became active in AARP. I was especially interested in the rights of retirees and was an advocate for improved access to health care for the elderly. I didn't go to the doctor regularly because I felt good, but I wanted to know I could get good care if I needed it.

60+ years

I had a mild heart attack at age 74, but still didn't worry much about my health. My wife died a couple of years later of breast cancer, and I was diagnosed with prostate cancer at age 80. They didn't operate because of my age and heart condition.

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Shirlene Hvinden
I was born in a little town in eastern Minnesota of Scandinavian parents. I have three older brothers and one younger sister. We lived a pretty normal middle-class lifestyle: My father worked as a school teacher during the year and as a used car salesman during the summers, and my mother stayed home and took care of us. My grandfather died of skin cancer when he was pretty old and my mother's sister died of breast cancer.

0–19 years

I loved to read as a child and did well in social studies and spelling, but poorly in math and science. I also loved to play and, despite my fair skin, spent most of my summers outdoors. I had allergies so I took medications regularly and saw the doctor often.

My mother cooked good, substantial meals (meat and potatoes) and I ate well, though I didn't like vegetables and wouldn't eat a bite more than the "spoonful" my mother made me eat of any vegetable but corn. After I graduated from high school, I went to college in St. Cloud, Minnesota.

20–39 years

When I was 21, I finished my education as a nurse, got married, and took a job at a community hospital in the outskirts of St. Paul. I had a son at age 22, then went on birth control pills until I was 28. Although I spent lots of time at the hospital, I rarely went to the doctor myself. I enjoyed my job, didn't drink or smoke, and certainly didn't experiment with drugs or do anything dangerous.

40–59 years

My life during these years was quiet and peaceful. My son lived at home as a college student, so I saw him often. I worked full time and contented myself with keeping house, sewing, and reading. I also began to volunteer one Saturday a month teaching teenage mothers how to care for their newborn children at a health care clinic in downtown St. Paul.

60+ years

When I was 61, I was asked to participate in a PLCO (prostate, lung, colon, ovarian) screening trial sponsored by the National Cancer Institute and administered through a local hospital. I was put into the group that received screening. The tests revealed that although my lungs and ovaries were okay, there were six large polyps in my colon. One of them appeared to be invasive, and the doctor ordered immediate surgery and chemotherapy. I did well for a time, but four years later, a routine check-up revealed more cancer.

Shirlene died at age 66 from colon cancer. She was survived by her husband and unmarried son.

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Clarence Robinson
I was born in Nashville, Tennessee, of African-American parents. My parents worked hard, but were poor and struggled to make ends meet for my two older sisters and one older brother. My father and his brother both died of lung cancer in their early 60s.

0–19 years

I was a pretty good student, but I loved music more than anything. I had a natural ear and could play both the guitar and the piano before I was 8. My talent and hard work earned me a spot in a band when I was12. By the time I was 16, I had quit school to go on the road. That was a good time, but tough—we ate and slept as best we could in those days, often grabbing any food we could find on the road. I also started smoking and drinking when I was 15 and didn't really start taking care of myself until I joined the army at age 18.

20–39 years

I stayed with the army till I was 25, then left and continued with my music, traveling for many years with a couple of the big bands. I didn't go to the doctor much, but, then, I didn't need to go. I always was pretty skinny. I guess living on cigarettes, alcohol, drugs, and women doesn't really promote gaining weight.

I don't think I have any children . . .

40–59 years

I developed a persistent cough in my early 40s but ignored it, figuring it was natural given my age and lifestyle. By now, I had stopped traveling and had settled down in a small apartment in Nashville, working as a back-up musician, mostly on piano. I never married, but with steady work and not traveling, I started eating better and getting more regular sleep.

60+ years

When I was 62, I gave up the lease on my apartment and moved in with my older sister. I had developed mild tremors by now and it was getting harder to play the piano. My cough continued—if anything, it was getting worse. When I was 67, the doctor told me I had mild emphysema. An X-ray at age 69, though, showed significant and inoperable lung cancer.

Clarence died at age 70 of lung cancer. He was survived by two older sisters and one older brother.

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Sami Khalafa
I was born in San Francisco in 1926, of an African-American father and Japanese mother. My family lived moderately: My parents were not rich, but they provided me with everything I needed and then some. My mother developed Alzheimer disease when she was in her late 60s, but there was no history of cancer in my family.

0–19 years

I was not a great student. Education was important to my mother, however, so I tried very hard. My father died of a heart attack when I was 15, so my mother and I went to live with my mother's sister. Our lifestyle didn't change much—we still ate mostly an Asian diet and our lives revolved around family activities.

When World War II started, my mother and her sister were sent to Utah to an internment camp for Japanese nationals living in the United States. I went with my mother and continued my schooling. It was there that I met the young Japanese man who was later to become my husband.

20–39 years

After the war, we moved back to San Francisco, where my mother began to take in laundry to support us. Soon after our return to the city, I was married, and my husband and I worked with my mother to build a good laundry and tailoring business. I was in good health except for a little arthritis from the regular sewing.

We had three children, but one died in infancy. We thought it was SIDS, but the doctors later said that it was thalassemia. After my children were born, we began to eat a more "Western" diet of meat and potatoes.

40–59 years

Our children both went to college. Our son became a successful businessman; our daughter went to law school and works as a defense lawyer in New York City. I worked less in the business and spent my time offering workshops at the community center on Japanese culture and language. It was important to me to promote an understanding of Japanese culture among people of all ethnic backgrounds.

60+ years

I was shocked when I was diagnosed with Parkinson disease at age 62. I had been active and healthy all my life, needing little in the way of health care. The Parkinson disease, on the other hand, took years to control. During that time, I saw my doctor regularly. When I was 67, she convinced me to have a sigmoidoscopy (an examination of my colon); it revealed three polyps, one of which had invaded other tissues. They removed the cancer surgically, but my relief at that was clouded by the Parkinson disease, which was getting worse.

Sami died at age 69 of Parkinson disease. Her husband and children remember her each year with a special family dinner on her birthday.

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Frank Trueblood
I was born in 1930 on a reservation in Wyoming, of Native American parents. I don't know of anyone in my family who had cancer.

0–19 years

I always hated school and did everything I could to avoid going. Our family was poor, but so was everyone else on the reservation, and somehow we got along. I don't remember going to the doctor much as a child, though when I broke my arm, my parents took me to the clinic for treatment.

To get off the reservation, I joined the army in 1947 (I lied about my age). It was in the army that I started drinking and smoking.

20–39 years

I moved back to Wyoming after I was discharged from the army. By then, I had decided that I wanted to be a rancher, and I went back to school on the GI bill to get a degree in agriculture. I did the rodeo circuit for a while when I was in college and after, but after a few more broken bones, I gave it up for full-time ranching.

40–59 years

Life on the ranch was good—I ate well (lots of meat and black coffee) and drank hard with my men. I never married (it was no life for a lady), though I have two kids who live in the city with their mother. It's just as well the kids don't live with me. Way out on the ranch, there's not much for them to do and there're no doctors and only a little local school.

I sometimes worry that I don't have a family to take care of me if I get hurt, but there's really nothing I can do about it. I don't even have health insurance because I can't afford it. I guess it's okay. I don't trust doctors anyway. The last time I saw one, he told me I had to quit drinking because I was developing liver problems. I ignored him; I've known plenty of men who've enjoyed booze all of their lives and still lived to a ripe old age. So what does he know?

60+ years

I developed a little abdominal pain after I passed 60, but I treated it with antacids and it always seemed to settle down afterward. One day, though, when I was 67, I got kicked by a horse and had to go to the hospital. They noticed blood in my stool and insisted that I get a test for colon cancer. Sure enough it was there, and before I knew it, they had cut me open and removed it. I went for chemotherapy for a while, but I'm okay now. Even the doc says I'm probably going to make it. The doc also says he can help me stop drinking. I'm lucky to get a second chance, so maybe I'd better try.

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Angela Seader
I was born in 1940 in St. Louis in a middle-class, African-American family. My parents were healthy most of their lives. As far as I know, there was no history of cancer in my family.

0–19 years

I got a basic education but never was much interested in school and quit when I turned 16 to help out at home. I was a healthy child and never went to the doctor much. When I was 19, I got married and moved to Chicago with my husband.

20–39 years

I started to smoke a little after I was married, but I never smoked much (not like my husband who smoked three packs a day). I had three children, though one died at birth. After the birth of my last child, I gave up smoking completely. I guess I figured I inhaled enough smoke with my husband's habit.

My husband had health insurance through his work, so my kids got good health care. My youngest had asthma, so we were at the doctor's often with her.

40–59 years

I went through menopause at about age 55, and because I was concerned about osteoporosis, I went on hormone replacement therapy right away. I didn't have any serious health problems except for occasional upper respiratory infections, a chronic cough, and frequent bronchitis (I was always sure the problem was the secondhand smoke from my husband's cigarettes). I had a mammogram every year and generally tried to keep in shape. I really wanted to live to see my grandchildren!

60+ years

When my husband died of lung cancer (he was 72), I was really glad I quit smoking. But at age 76, the doctor made me get a chest X-ray to see what the cause of my chronic cough was. He discovered a massive tumor in the middle of my left lung. It was too big to operate, but I did have chemotherapy. Eventually, I had to use oxygen in order to breathe easily. Finally, one of my upper respiratory infections turned into pneumonia.

Because of my advanced lung cancer, I decided to refuse treatment with antibiotics.

Angela died three days after being hospitalized with pneumonia. She was survived by six grandchildren.

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Sam Major
I was born in downtown Chicago in 1939, of African-American parents. I was the middle child of three. My parents both smoked, but there wasn't any history of cancer in the family.

0–19 years

I was an okay student and finished high school with no problems. I was sick a lot as a child, but mostly it was just little stuff, like colds and the flu and such. Seemed like I was always on antibiotics as a child though.

I was a picky eater. Mostly, I liked really basic food. For sure, I didn't like eating vegetables!

I started smoking when I was 17, but I never had what you'd call a bad habit. Maybe three-quarters of a pack a day, maybe a pack now and then. Nothing really big.

20–39 years

I moved to Milwaukee when I left home and became a salesman. I got a little college under my belt by going to night school and eventually got to be in charge of auto parts sales in a pretty large district. By then, I was married and had a son, but I didn't see my family too much because I was traveling three and four days each week.

40–59 years

I slowed down a little as I got older and picked up some hobbies—a little wood carving and some reading. I was still traveling, though, which was hard on my wife. How she put up with me for 32 years, I'll never know.

When I was 57, I started feeling pain in my abdomen and finally went to the doctor. He thought it might be a bladder infection and put me on antibiotics, but they didn't work. Finally, they did a test and found a tumor in the lining of my bladder. The tumor was pressing against my urethra, and that was what was causing the pain. They had to take the bladder out and put in a pouch for my urine that I have to drain by hand. They also put me on both chemotherapy and radiation therapy. It was a tough time.

60+ years

I'm 60 now and doing just fine. I still see the oncologist regularly, and he makes sure I'm taking good care of myself. Last year, I even quit smoking, at his insistence. Next thing you know, I'll be eating my vegetables!

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Eleanor Hartman
I was born in 1933 in Oregon of white, middle- to upper-class parents. We had a big home outside the city, and I went to a private school just up the lane. There wasn't any history of cancer in my family.

0–19 years

I was an okay student in school—Bs and sometimes a few As. I don't remember having any serious health problems. I had my tonsils out at 8, and I had two broken arms from falling off my horse. Other than that, my childhood was uneventful. We were a pretty traditional family, you know, meat and potatoes for dinner, no smoking, only social drinking, and that not very often.

20–39 years

I went to college right out of high school, but quit after three years to get married. My husband owned a small accounting firm, and I was a housewife. We had one daughter. I raised her and spent the rest of my time volunteering at her school and at the local hospital.

My health was pretty good during these years. I was sometimes tired, but I took iron and tried to get lots of rest. My husband and I didn't go to the doctor much because we were trying to keep our health insurance costs as low as possible. (When you're self-employed, health insurance costs can be very high.)

40–59 years

My daughter got married and moved away, and I filled my time with more volunteer work. By now, my husband's company had grown much larger, and he was busier than ever before. I was glad he was successful, but pretty lonely. I had my gall bladder out when I was 48, but other than that, I had no serious health problems.

60+ years

I started slowing down after I passed 60. My husband wanted to retire, and we both wanted to travel. But I started having abdominal pain, and one day, I noticed that I was bleeding from my rectum. I went to the doctor right away and learned I had pretty advanced colon cancer. Talking to the doctor, I realized that the periodic diarrhea that I had in my late 40s and 50s probably was related to the cancer, but how was I to know? I thought it had to do with losing my gall bladder!

They operated to remove as much of the tumor as possible and placed an internal arterial pump to deliver chemotherapy to my liver to help control the cancer that had spread there. Six months later, a scan showed more tumors in my liver. The doctor told me there is nothing more they can do except try to control the symptoms. She estimates that I have six months to a year to live.

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Christopher Bartling
I was born in Memphis in 1920, the second of six children of African-American parents. We were pretty poor in money, but rich in love. My father died of prostate cancer when he was 82.

0–19 years

We were a happy family. We worked hard and played hard and ate well (lots of barbequed beef and pork, heaps of french fries—the works!).

Despite my parents' love and attention, I grew up a little wild. I finished high school, but I started drinking when I was a teenager and never really stopped. I got a job as a laborer on the railroad after I graduated from high school and never worked for anyone else.

20–39 years

I settled down outside of Memphis and had a comfortable life. I still drank pretty heavily on the weekends, but it never interfered with my working. In fact, by now, I was making my own moonshine liquor and handing it out or selling it to my friends. I almost never went to the doctor and probably didn't eat all that well, living alone as I did. My friends used to say I needed a wife to take care of me, but I liked the bachelor's lifestyle.

40–59 years

When my mother died of a heart attack (I was 42 at the time), I felt kind of alone in the world. I had brothers and sisters, but didn't see them often. So one day, I decided to get myself a dog. It was a good thing—the dog kept me company on the weekends and waited patiently all day for me to come home and play with her after work. I never was much for exercise (as a laborer, I figured I got plenty when I worked), but I started taking long walks with the dog. Those were good times. The doctor even says that's probably why my heart is so strong.

60+ years

I retired from the railroad the day I turned 65. Retirement was pleasant—I spent most of my days puttering in my garden and playing with my still and experimenting with different kinds of brews.

They found the cancer when I was 75 years old. I started to lose weight and had some pains in my abdomen, so I went to see the doctor. She said I had pancreatic cancer, pretty far along, and that there wasn't much they could do for me. They wanted to put me in a nursing home, but I told them they could keep their nurses and their treatments: I was going home. I had a good life and if it was time to go, that's the way it was.

Christopher died eight months after being diagnosed with pancreatic cancer. He was 76.

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Jonah Wexler
I was born in 1983 in Los Angeles, of middle-class Caucasian parents. There was no history of cancer in my family.

0–19 years

After a routine pregnancy and delivery, I developed normally for the first few months of my life. My mother took me for regular check-ups and immunizations.

Because I was a first child, I got lots of attention. My caregivers noticed early on that my eyes were different colors (one was blue and the other hazel), but the doctor said not to worry. A few months after my first birthday, my mother noticed my left eye was always red and I seemed to be squinting. In response to her concern, my doctor examined me more closely and sent me to an opthamologist for a complete examination. This exam revealed that I had retinoblastoma, a form of cancer that develops inside the eye. I was treated with surgery and radiation and within a few months was pronounced "cured" and "doing fine."

The rest of my childhood was uneventful.

20–39 years

I went to college right after high school and eventually graduated with a degree in biology. I went on to study genetics in graduate school and finally earned my certification as a genetic counselor. I was married a couple of months afterward, and my wife and I moved to Philadelphia, where I got a job as a genetic counselor.

My wife and I were unable to have children, so we adopted two, a boy and a girl.

40–59 years

After our children went away to college, my wife and I sold our home and moved into an apartment overlooking the river in Philadelphia. I continued my work in genetics, now as director of a major center for genetic counseling and testing. When I counsel parents whose children have retinoblastoma, I often think back to how scary it must have been for my parents to have an infant with cancer. I try to help my clients as much as someone long ago must have helped my parents.

60+ years

Although I've enjoyed my career in genetic counseling, I have to admit that I'm looking forward to retirement. I am grateful that I have remained cancer-free since my first bout with it as an infant, but I am not naive: I get regular check-ups and watch for changes that could signal another problem. There's no doubt in my mind that early detection and treatment is a key part of surviving the disease.

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Mary Snyder
I was born in 1950, of Caucasian parents living in a suburb of Chicago. My father was a math teacher in the local high school, and my mother worked as a nurse. There wasn't any history of cancer in my family.

0–19 years

I was an okay student in school, though I never liked it much. My parents divorced when I was 11, and my mother and I moved into the city. She continued working as a nurse, but also went back to school to get her master's degree. She wasn't around very much those days—I guess she was dealing with her own problems—and I was alone a lot. I started hanging out, began smoking and drinking, and finally, in the middle of my junior year, I dropped out of school.

I became sexually active when I was 14, and though I tried to get the guys to use condoms, often they didn't. Because I was scared I'd get pregnant, I finally went to the local clinic and got birth control pills. I still worried though, about what I might catch from a guy.

20–39 years

When I was 21, a girlfriend and I got an apartment together, and I started working as a receptionist at a car dealership. The health plan wasn't real good there, and I only went to the doctor when I needed another prescription for birth control pills. When I was 26, a routine Pap smear showed some abnormal results. When I went back to be checked again, it was normal. I figured the doctor must have messed up and didn't go back for my next check-up.

I finally went for another check-up when I was 33. This time, the results were clear: I had cervical cancer. I was pretty scared and called my mom to ask her about it. She said that this type of cancer has a high survival rate if it is caught early. They removed the cancer and gave me some literature about risk factors associated with cervical cancer. I learned that condoms protect against more than just pregnancy, and promised myself that in the future, I'd always have a supply handy.

40–59 years

Since my experience with cancer, I've become more careful about lots of things in my life. I'm eating a better diet, I stopped smoking, and now the guys I see always wear condoms. I'm even thinking of going back to school.

60+ years

With retirement just around the corner, I've decided to move out of the city back to the little suburb where I was born. I'm thinking I could get a little apartment overlooking the park where my dad and I used to walk the dog, and maybe I can volunteer in the local school.

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Shaun O'Malley
I was born of Irish-American parents in 1936 in western Pennsylvania. My dad worked in the steel mills, and my mom ran a little coffee shop. Both my parents had skin cancer before they were 60.

0–19 years

I was a good student in school but always preferred to be outdoors rather than cooped up inside doing homework. I loved any type of sports, especially baseball. When I was in high school, I started working on a construction crew in the summer. Because of my fair skin, I got lots of bad sunburns, but I didn't really care.

20–39 years

I continued working in construction after I finished high school. Soon I was a foreman and making enough money to get married. My wife and I both enjoyed socializing; on Friday nights, I especially enjoyed hanging out in the local bar where I would play darts and watch TV.

We had two kids, both with fair skin and freckles, just like their dad.

40–59 years

I started gaining weight when I was in my 40s, not bad, but a little, and my wife started nagging me about seeing a doctor. When I was 45, to make the wife happy, I finally went for a check-up. All the doctor found was that my blood pressure was a little high. She gave me medication for that and cautioned me to continue getting my exercise.

When I was 57, my wife started nagging me again, this time about some moles and freckles on my neck and shoulders that she thought were suspicious looking. I went back to the doctor again, but this time, she referred me to a dermatologist. Sure enough, my wife was right: Several of them were cancerous and had to be removed.

60+ years

Now that I understand about skin cancer, I go for regular check-ups. So far, I haven't had another problem, but I'm not taking any chances. I also have started nagging my children about wearing sunscreen and about seeing that their children do too.

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Paul Ashland
I was born in 1924 in northern Michigan of African-American parents. My older sister had lung cancer when she was in her 60s, but she was a smoker, so we weren't surprised.

0–19 years

We had a normal childhood: My father worked, and my mother stayed home to watch the kids. We ate well—my father especially loved steak and baked potatoes for dinner—but I wasn't overweight because I was active in sports. I started chewing a little when I was 18 (I also started drinking a little—all the guys did it).

20–39 years

After I finished high school, I got a good job with a trucking company and started making long-distance hauls with a partner. I was on the road a lot, so I didn't really develop any hobbies or outside interests. We traveled five days out of seven, and slept and ate on the road. Chewing helped me keep awake on long hauls.

40–59 years

By the time I was in my mid-40s, I started gaining weight. When I developed headaches, I went to the doctor to see what was wrong. He said I had high blood pressure, but said I could control it with diet if I tried to. I lost some weight on the diet he prescribed, and the headaches went away. I'm usually pretty good about sticking to the diet, though I do like a drink or two after a long day on the road.

60+ years

I retired when I was 65. Retirement was hard on me: I was used to traveling and didn't really have friends except for other truckers. To ease my loneliness, I hung out at the terminal and helped load and unload the trucks just for the heck of it.

When I was about 69, I noticed soreness in my mouth and saw something that looked like a large canker sore. I figured it would go away. It didn't. Then I noticed a lump underneath it. It was pretty sore, so I decided to see a doctor. She took a biopsy and found that I had throat cancer. The surgery was tough, and I don't look the same. I don't go out much now, even to the docks. My mouth is dry and sore all the time from the radiation. Between that and the chemotherapy, I really can't eat much and don't taste what I do eat. I continue to lose weight and feel bad most of the time. I really miss seeing the guys from the docks.

Paul died at age 71 of cancer. Twelve of his buddies from the trucking company attended his funeral.

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Sharon Washington
I was born in 1938 in rural Vermont of Caucasian parents. We lived in an expensive neighborhood (my father worked as a chauffeur for a wealthy businessman), but we were always pretty poor.

0–19 years

I was an excellent student in school. My mother taught me to read when I was very young, and I read everything I could get my hands on—historical novels, science fiction, poetry. We couldn't afford to buy many books, but that was okay: There was a little public library just up the road that I could ride my bike to, and I spent lots of happy hours there, reading and dreaming of the day I would have my own library.

When I finished high school, I decided I wanted to become a librarian. With my parents' encouragement, I applied to several colleges. When one accepted me and offered me a job so I could work for my tuition, I moved away from home, promising my folks that one day they'd be proud of me.

20–39 years

It took me six years to get through college because I had to work for my tuition, but it was worth it. After I graduated with a degree in library science, I moved back to New England to be near my folks and to work as a reference librarian in a small college library. I had a good life; though I never married, I stayed active socially and enjoyed gardening, skiing, hiking, and, of course, reading.

40–59 years

In my early 40s, I had some problems with migraines and depression; the doctor prescribed an antidepressant. Other than that, I was in good health till my late 50s.

Just after I turned 58, I started feeling some pain in my abdomen. I ignored it until the bloating got so bad that it was interfering with my gardening. When I went to the doctor, she sent me for tests immediately and discovered that I had ovarian cancer. The oncologist operated, then put me on chemotherapy but said that we caught it so late that I had only a 25 percent chance of recovery.

60+ years

Sharon died at age 61 of ovarian cancer. Her will directed that her large personal library be donated to the little public library in Vermont where she spent so many happy hours as a child.

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Leon Sanchez
I was born in 1940, the third of four children of Hispanic parents. My parents were fortunate: They were healthy and independent (they owned their own small farm in western Alabama). The only serious illness in the family was when my father—and then, 20 years later, my older brother—were diagnosed with prostate cancer.

0–19 years

We lived a simple, uncomplicated life. We ate well (lots of meat and vegetables) and played and worked hard. My parents believed in education and insisted that we all finish school, whether we wanted to or not. I was not an honor student, but I got consistent Bs. Math was my best subject and history my worst.

20–39 years

As an adult, I continued working on my father's farm and eventually inherited it from him (my brothers and sister had moved away). I loved the outdoor life—it was hard work, but honest, and it gave me a good feeling to work the land I owned. And it produced a good living for my wife and two children. The only disadvantage of the farm was how far we were away from the town and a doctor (especially if we needed one fast).

40–59 years

After my children moved away (one to college, the other to start his own landscaping business in the suburbs of Birmingham), my wife and I became even more isolated, going into town only once a month or so for supplies. We loved the quiet life on the farm and had few worries.

60+ years

By the time I was 60 or so, I was beginning to feel my age. Finally, I decided it was time to see a doctor for a check-up. I wanted to make sure that I was in okay health, and I wanted to ask him why I was starting to feel pain when I urinated.

The doctor checked me over and pronounced me healthy in all regards except one: Tests revealed that, like my father and older brother before me, I had prostate cancer. Because of my generally good health, they were able to operate, and then they treated it with radiation. I don't keep up much with modern medicine, but whatever they did, it must have worked, because the pain is gone now and I feel better than ever.

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Marcy Sterling
I was born in Florida in 1942, the oldest of two girls born to Scandinavian parents. I think my grandmother died of colon cancer, but I don't think anyone else in my family had cancer.

0–19 years

I had a wonderful childhood. My sister and I loved the beach and adored being outdoors—Florida was a great place for an active life. We went sailing almost every weekend. And we spent many, many hours playing on the beach and in the water. Even the sunburns we kept getting (our fair skin never tanned) didn't discourage us—as soon as we were healed, we'd be back outside.

I was a good student and went on to college after high school. My goal was to become an elementary teacher (preferably a kindergarten teacher).

20–39 years

After college, I got a job teaching fourth grade in an elementary school in Miami. After my practice teaching, I had decided that I really enjoyed the older kids more than the younger ones, and fourth to fifth grade seemed just right. I enjoyed teaching, and I think I was pretty good at it. I still loved the outdoors and took every opportunity to be out in the sun, whether during the week or on weekends.

As I got older, I began to watch my health and especially my skin more and more. I read about the link between sunburns and skin cancer and that worried me. I married a physician, and he insisted that I use sunscreen regularly and remember to wear a hat outdoors. I still loved sailing and going to the beach (so did he), but now I was careful to guard myself and our little son against getting burned.

40–59 years

Thanks to my husband's concern, I saw my doctor for annual check-ups and followed her instructions religiously. I developed slightly elevated blood pressure as I aged, but the doctor found a combination of medication and diet that controls it, so I didn't worry about it.

Because of my history of sunburns, she also checked me carefully for any changes in moles or any other signs of skin cancer. Sure enough, when I was 56, she found a few that were beginning to change. Eventually, I had three of the moles removed surgically. Both the oncologist who did the surgery and my regular doctor said that because we caught the skin cancer early, my chances of recovery were very good.

60+ years

My husband and I are both retired now, and tennis, golf, sailing, and the beach are still regular parts of our lives.

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Shawna Thomas
I was born in a little town in Louisiana in 1950 of African-American parents. As far as I know, there was no history of cancer in my family.

0–19 years

My father had been in the army before I was born. When he came home, he went to college on the GI bill. After college, he became a history teacher in the local high school. We lived in a small but nice house on the outskirts of town. My mother had a garden and raised chickens, so we ate lots of vegetables and eggs.

I had asthma as a child, so I saw the doctor regularly. Other than that, my childhood was uneventful. I was a good student and dreamed of becoming a doctor myself.

20–39 years

I worked hard in college and, after several tries, was admitted to a medical school for women in Philadelphia. After I finished my training, I returned to Louisiana to work in a clinic near the little town I grew up in. When I was 30, I married a local businessman and we started a family. I had two children.

Because of my medical training, I was very careful about watching my body for any changes. One morning when I was 38, I discovered a spot in my left breast that just didn't feel right. I went immediately to my doctor, and he sent me for further testing. Sure enough, it was a small breast tumor. I had a partial mastectomy (in those days, they often removed more tissue than they had to), followed by chemotherapy.

40–59 years

After I recovered from the cancer, life was different for me. The shock of being ill had caused me to re-evaluate my priorities. I reduced the number of hours I was spending in the clinic, preferring to spend more time at home with my husband and children. I regularly examined my remaining breast tissue and also went for regular cancer check-ups. Fortunately, we had caught the cancer before it spread, and five years after my surgery, I was still cancer-free.

60+ years

My children are grown, and I am retired now, preferring to volunteer instead of work for pay at the small hospital the town finally built. My husband died last year of a stroke, and I decided to offer a scholarship in his name each year to a young person from the area who wants to go on to college. Life is good. After all these years, I have finally lost my fear that the breast cancer will return (but I still see my doctor regularly).

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Team Summary

Use the information provided in your team's identity envelopes to complete this worksheet.

Section 1: Family History
Tally the number of people in your team

- who have a history of cancer in their family _____
- who do not have a history of cancer in their family _____

Section 2: Cancer History
Complete the table by writing in the number of people in your team who were diagnosed with cancer during each period of life. Then list the type of cancer each person developed. If no one was diagnosed with cancer, leave the section blank.

Team's Cancer History

Type of Information	Period of Life			
	0–19 years	20–39 years	40–59 years	60+ years
number of people diagnosed with cancer				
type of cancer				

Section 3: Possible Risk Factors
Go back through your cards and identify possible risk factors associated with the development of cancer in the people in your team. List those risk factors here.

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Drawing Conclusions from the Faces of Cancer

Complete this worksheet as your class compiles the data from the *Team Summaries*.

Conclusion One: Family History

Conclusion Two: Relationship Between Cancer and Age

Conclusion Three: Types of Cancer

Conclusion Four: Possible Risk Factors

Discussion Questions

1. In this activity, all students in the class assumed the role of someone who developed cancer sometime in his or her lifetime. Is this an accurate representation of the risk of cancer among the American population? Explain your answer.

2. What explanation can you offer for the observation you made about the incidence of cancer compared with age?

3. What is the most interesting or surprising thing you learned from this activity? What is the most important? Why?

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Summary Profile of the Faces of Cancer

Type of Information	Accumulated Class Data			
family history (write in the number of "yes" and "no" answers for all teams)	yes _____ no _____			
	Period of Life			
	0–19 years	20–39 years	40–59 years	60 and older
number of people diagnosed with cancer (write in the total for all teams)				
type of cancer (write in the number of each)	bladder ____ brain ____ breast ____ cervical ____ colon ____	leukemia ____ lung ____ oral cavity ____ ovarian ____ pancreatic ____	prostate ____ retinoblastoma ____ skin ____ uterine ____ other ____	
possible risk factors (list any possibly relevant factors)				

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Understanding Cancer

Use the resources provided on the CD-ROM to complete this worksheet.

Section 1: Factors Reported to Be Associated with Cancer
View the *News Alert* videos and use the information provided to identify what each video suggests is the cause of cancer and what evidence supports that claim.

***News Alert* Video**	**Factor Proposed to Cause Cancer**	**Evidence**
Cancer and Chemical Poisons		
Cancer and Your Family History		
Cancer and Radiation Exposure		
Cancer and UV Light		

Section 2: Building an Explanation for the Cause of Cancer
View the animations on the CD-ROM. Think about the information each animation presents, then write a one-sentence statement for each that summarizes what you learned.

Animation 1:
Cancer involves . . .

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Animation 2:

Cell division normally is . . .

Animation 3:

Cell cycle regulation is accomplished by . . .

Animation 4:

Cancer-causing agents often . . .

Animation 5:

When damage occurs to genes that regulate the cell cycle . . .

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Section 3: Explaining Factors Associated with Cancer

Review your notes from Section 1, then write a sentence that describes how our current understanding of cancer explains the role that each factor plays in causing cancer.

Cancer and Chemical Poisons

Cancer and Your Family History

Cancer and Radiation Exposure

Cancer and UV Light

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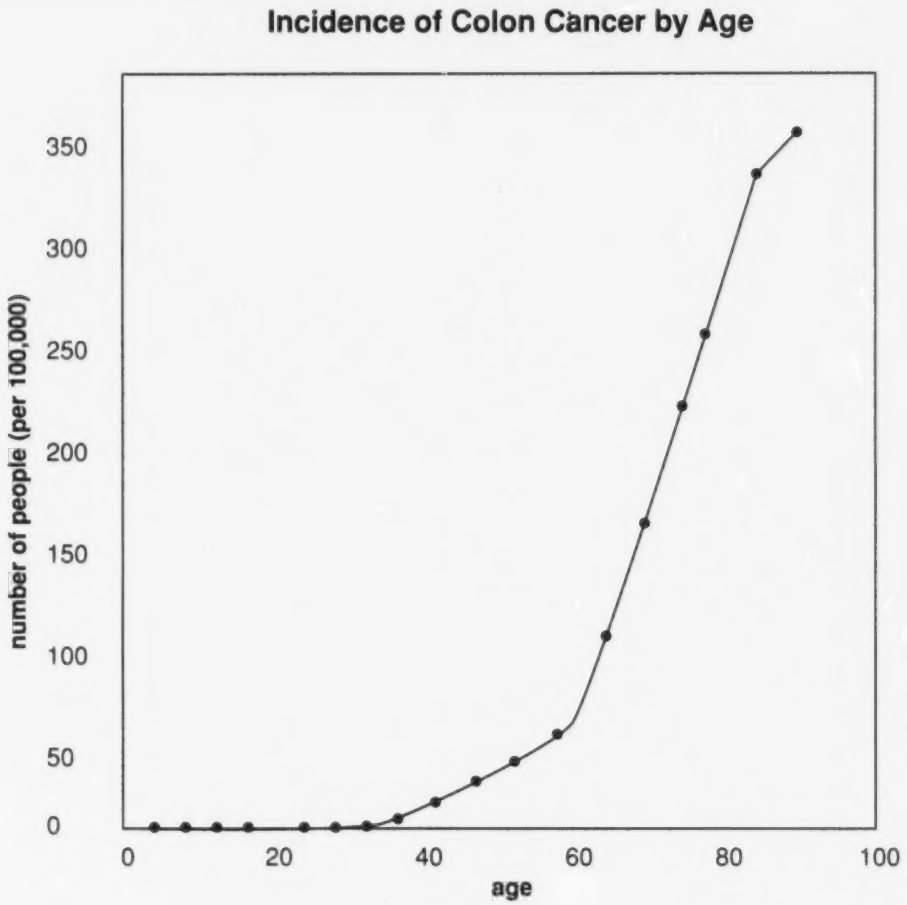
Colon Cancer Incidence by Age

Examine the following graph, then answer the questions.



1. How likely is it that you will develop colon cancer this year?

2. How likely is it that someone who is 60 years old will develop colon cancer this year?

3. How likely is it that someone who is 80 years old will develop colon cancer this year?

4. How can we explain this change in the risk of a person developing colon cancer?

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Random Number Tables

Student 1		
Age	**Gene 1**	**Gene 2**
5	18	21
10	15	3
15	12	2
20	6	24
25	24	16
30	16	15
35	18	8
40	24	12
45	11	16
50	21	17
55	23	4
60	14	2
65	10	10
70	17	16
75	7	16
80	2	19
85	16	24
90	16	21
95	1	18
100	24	11

Student 2		
Age	**Gene 1**	**Gene 2**
5	18	21
10	18	16
15	11	8
20	12	23
25	18	8
30	4	2
35	24	5
40	24	18
45	14	3
50	18	10
55	4	21
60	11	25
65	19	21
70	5	20
75	5	14
80	3	2
85	18	21
90	1	8
95	1	2
100	5	13

Student 3		
Age	**Gene 1**	**Gene 2**
5	24	22
10	4	17
15	23	23
20	24	5
25	4	1
30	13	7
35	16	22
40	11	17
45	20	1
50	10	17
55	16	11
60	8	18
65	3	5
70	7	8
75	16	9
80	12	14
85	11	3
90	24	16
95	4	17
100	25	3

Student 4		
Age	**Gene 1**	**Gene 2**
5	16	18
10	4	13
15	2	15
20	12	10
25	5	17
30	8	3
35	2	1
40	6	11
45	24	23
50	2	7
55	24	5
60	21	19
65	10	18
70	24	21
75	22	11
80	16	20
85	20	1
90	18	15
95	7	11
100	21	4

Student 5		
Age	**Gene 1**	**Gene 2**
5	6	21
10	12	13
15	14	7
20	21	22
25	15	10
30	17	10
35	23	16
40	25	14
45	13	11
50	4	25
55	20	23
60	13	16
65	24	24
70	15	5
75	19	14
80	17	25
85	25	22
90	16	17
95	25	22
100	2	3

Student 6		
Age	**Gene 1**	**Gene 2**
5	23	12
10	11	9
15	13	8
20	5	3
25	14	25
30	16	15
35	2	20
40	22	24
45	5	8
50	18	16
55	6	5
60	13	20
65	2	1
70	2	7
75	1	2
80	14	22
85	10	9
90	4	20
95	18	22
100	17	20

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Student 7		
Age	**Gene 1**	**Gene 2**
5	6	8
10	5	19
15	25	8
20	5	18
25	18	23
30	7	7
35	16	6
40	9	21
45	11	15
50	19	23
55	15	21
60	21	18
65	21	11
70	19	20
75	24	7
80	19	23
85	19	22
90	1	13
95	18	14
100	10	20

Student 8		
Age	**Gene 1**	**Gene 2**
5	2	1
10	25	25
15	5	22
20	7	14
25	25	20
30	20	18
35	12	22
40	11	3
45	23	8
50	7	1
55	14	5
60	9	21
65	3	9
70	9	4
75	17	16
80	1	19
85	5	25
90	24	1
95	13	20
100	11	21

Student 9		
Age	**Gene 1**	**Gene 2**
5	19	14
10	2	25
15	10	12
20	5	17
25	19	4
30	23	22
35	1	6
40	6	9
45	5	4
50	21	25
55	25	24
60	5	9
65	4	7
70	23	11
75	14	13
80	2	14
85	18	25
90	19	6
95	4	6
100	4	1

Student 10		
Age	**Gene 1**	**Gene 2**
5	21	3
10	7	20
15	3	2
20	23	17
25	5	22
30	7	13
35	14	2
40	4	12
45	19	16
50	22	23
55	9	21
60	25	19
65	2	1
70	24	25
75	24	12
80	14	13
85	13	9
90	6	18
95	5	23
100	11	25

Student 11		
Age	**Gene 1**	**Gene 2**
5	3	7
10	21	17
15	23	16
20	6	14
25	13	5
30	18	21
35	12	18
40	19	15
45	24	6
50	17	24
55	13	14
60	13	20
65	18	20
70	18	3
75	2	25
80	12	10
85	8	25
90	18	7
95	22	18
100	13	21

Student 12		
Age	**Gene 1**	**Gene 2**
5	19	5
10	12	16
15	24	14
20	22	23
25	8	15
30	7	9
35	16	23
40	17	10
45	7	3
50	12	14
55	7	13
60	5	17
65	3	22
70	11	15
75	10	7
80	7	18
85	21	25
90	4	23
95	6	6
100	6	17

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Student 13		
Age	Gene 1	Gene 2
5	11	4
10	19	10
15	12	18
20	6	7
25	15	1
30	5	21
35	15	24
40	9	2
45	9	22
50	5	8
55	4	8
60	19	8
65	4	3
70	19	2
75	24	6
80	20	19
85	22	2
90	9	13
95	24	17
100	24	1

Student 14		
Age	Gene 1	Gene 2
5	2	23
10	13	19
15	22	16
20	17	6
25	7	4
30	17	23
35	12	6
40	7	7
45	10	5
50	7	20
55	18	25
60	22	10
65	9	22
70	10	17
75	4	1
80	10	8
85	9	23
90	4	24
95	6	17
100	21	12

Student 15		
Age	Gene 1	Gene 2
5	11	5
10	15	6
15	2	22
20	12	15
25	5	15
30	24	21
35	20	2
40	2	11
45	21	5
50	24	19
55	4	24
60	11	18
65	16	18
70	4	3
75	22	20
80	15	19
85	7	17
90	17	13
95	20	18
100	9	17

Student 16		
Age	Gene 1	Gene 2
5	22	6
10	16	7
15	4	6
20	25	1
25	4	7
30	1	6
35	10	15
40	10	12
45	2	10
50	12	14
55	2	22
60	17	2
65	15	17
70	19	13
75	22	18
80	22	12
85	14	12
90	12	17
95	12	17
100	5	22

Student 17		
Age	Gene 1	Gene 2
5	7	21
10	19	2
15	17	9
20	11	14
25	5	17
30	9	9
35	7	19
40	23	8
45	18	21
50	12	14
55	2	15
60	15	3
65	17	12
70	16	20
75	2	21
80	23	13
85	10	13
90	17	24
95	25	25
100	8	2

Student 18		
Age	Gene 1	Gene 2
5	2	13
10	18	15
15	11	17
20	4	11
25	24	20
30	14	14
35	8	4
40	1	8
45	20	13
50	9	17
55	14	8
60	20	12
65	19	16
70	16	16
75	5	15
80	16	3
85	14	13
90	12	21
95	5	4
100	3	10

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Student 19		
Age	Gene 1	Gene 2
5	1	21
10	13	18
15	11	4
20	15	4
25	2	8
30	22	8
35	19	8
40	22	25
45	18	23
50	12	20
55	23	24
60	16	5
65	2	3
70	2	24
75	1	5
80	21	5
85	15	4
90	10	10
95	3	24
100	17	7

Student 20		
Age	Gene 1	Gene 2
5	2	15
10	16	25
15	22	2
20	24	20
25	6	8
30	1	9
35	1	12
40	11	11
45	19	1
50	12	4
55	6	4
60	2	3
65	2	18
70	5	15
75	15	15
80	1	18
85	13	15
90	25	10
95	15	14
100	22	16

Student 21		
Age	Gene 1	Gene 2
5	3	21
10	22	3
15	7	24
20	7	13
25	13	13
30	21	24
35	18	22
40	18	14
45	24	6
50	1	8
55	14	18
60	18	24
65	25	2
70	24	3
75	2	6
80	7	22
85	24	14
90	22	15
95	8	17
100	5	4

Student 22		
Age	Gene 1	Gene 2
5	2	18
10	15	25
15	17	5
20	5	8
25	25	19
30	6	5
35	23	20
40	24	12
45	10	5
50	15	7
55	15	25
60	7	23
65	24	7
70	23	19
75	18	10
80	4	9
85	14	20
90	22	21
95	9	25
100	25	21

Student 23		
Age	Gene 1	Gene 2
5	24	17
10	11	15
15	13	24
20	8	14
25	2	25
30	12	10
35	20	12
40	14	13
45	10	4
50	8	23
55	17	10
60	6	3
65	13	19
70	25	9
75	9	17
80	14	1
85	13	1
90	20	18
95	22	5
100	13	1

Student 24		
Age	Gene 1	Gene 2
5	24	24
10	8	16
15	5	11
20	8	24
25	9	15
30	3	19
35	3	15
40	12	13
45	7	1
50	1	17
55	14	7
60	14	8
65	24	2
70	8	6
75	21	8
80	14	2
85	5	25
90	6	12
95	21	22
100	5	19

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Student 25		
Age	Gene 1	Gene 2
5	10	20
10	10	20
15	8	8
20	9	16
25	23	9
30	18	7
35	19	24
40	14	13
45	10	11
50	5	16
55	13	3
60	2	7
65	20	11
70	19	18
75	19	11
80	12	25
85	10	2
90	13	9
95	8	21
100	25	4

Student 26		
Age	Gene 1	Gene 2
5	2	7
10	4	6
15	10	9
20	18	23
25	3	1
30	15	10
35	8	10
40	7	12
45	14	8
50	10	15
55	18	25
60	19	6
65	11	23
70	5	18
75	5	13
80	15	21
85	13	18
90	12	14
95	17	6
100	14	15

Student 27		
Age	Gene 1	Gene 2
5	3	12
10	6	16
15	23	14
20	11	22
25	15	13
30	3	17
35	1	6
40	16	18
45	7	17
50	24	11
55	13	4
60	2	6
65	7	6
70	3	13
75	8	24
80	24	23
85	18	21
90	17	12
95	2	1
100	13	11

Student 28		
Age	Gene 1	Gene 2
5	17	2
10	15	7
15	1	18
20	4	15
25	21	20
30	21	9
35	3	25
40	14	14
45	1	7
50	17	10
55	1	19
60	21	8
65	15	15
70	12	11
75	15	14
80	2	7
85	9	2
90	19	19
95	21	3
100	21	25

Student 29		
Age	Gene 1	Gene 2
5	11	19
10	12	1
15	10	12
20	7	23
25	18	4
30	9	7
35	10	20
40	10	17
45	20	14
50	1	18
55	17	2
60	24	2
65	23	18
70	9	24
75	22	12
80	8	15
85	15	8
90	2	8
95	9	24
100	14	3

Student 30		
Age	Gene 1	Gene 2
5	6	22
10	20	15
15	10	10
20	20	25
25	23	7
30	6	25
35	5	3
40	24	5
45	8	7
50	25	18
55	23	1
60	20	10
65	19	25
70	10	25
75	23	3
80	22	6
85	2	15
90	8	2
95	9	23
100	25	11

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Student 31		
Age	**Gene 1**	**Gene 2**
5	19	16
10	20	21
15	12	22
20	21	14
25	6	5
30	23	18
35	20	2
40	25	4
45	22	20
50	7	15
55	10	6
60	20	14
65	4	12
70	7	16
75	2	9
80	25	12
85	14	4
90	4	9
95	23	22
100	6	6

Student 32		
Age	**Gene 1**	**Gene 2**
5	2	9
10	18	25
15	11	23
20	20	23
25	12	16
30	1	12
35	3	1
40	22	11
45	4	19
50	11	5
55	2	24
60	8	19
65	9	18
70	1	14
75	10	5
80	16	15
85	22	3
90	10	20
95	1	5
100	23	9

Student 33		
Age	**Gene 1**	**Gene 2**
5	3	21
10	25	11
15	22	12
20	23	17
25	24	8
30	15	16
35	22	14
40	24	24
45	13	18
50	20	17
55	8	23
60	13	22
65	20	16
70	16	11
75	18	10
80	4	21
85	10	6
90	24	12
95	18	16
100	24	23

Student 34		
Age	**Gene 1**	**Gene 2**
5	22	10
10	2	24
15	13	18
20	19	24
25	24	4
30	1	6
35	16	9
40	25	24
45	2	16
50	15	14
55	12	20
60	2	22
65	8	23
70	24	12
75	13	23
80	8	16
85	7	1
90	2	13
95	12	9
100	19	4

Student 35		
Age	**Gene 1**	**Gene 2**
5	5	2
10	19	1
15	18	18
20	19	20
25	14	8
30	15	4
35	4	25
40	24	22
45	7	2
50	8	23
55	12	13
60	14	23
65	10	4
70	4	25
75	5	12
80	12	11
85	18	14
90	25	20
95	1	22
100	3	25

Student 36		
Age	**Gene 1**	**Gene 2**
5	12	19
10	21	14
15	8	19
20	16	24
25	24	24
30	18	20
35	6	2
40	4	2
45	10	2
50	7	25
55	3	4
60	16	9
65	25	17
70	6	24
75	20	7
80	4	25
85	19	13
90	14	17
95	9	22
100	20	1

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Student 37		
Age	Gene 1	Gene 2
5	6	14
10	15	22
15	8	25
20	18	17
25	25	24
30	12	8
35	11	22
40	7	17
45	7	3
50	10	2
55	16	23
60	8	19
65	6	7
70	5	6
75	22	14
80	8	14
85	3	4
90	22	10
95	5	16
100	14	17

Student 38		
Age	Gene 1	Gene 2
5	2	24
10	9	24
15	6	10
20	16	10
25	18	14
30	20	20
35	10	3
40	15	22
45	2	20
50	14	24
55	8	25
60	4	1
65	23	17
70	15	1
75	6	9
80	3	4
85	13	4
90	7	8
95	9	19
100	1	2

Student 39		
Age	Gene 1	Gene 2
5	1	12
10	18	14
15	23	22
20	14	19
25	18	3
30	13	16
35	2	23
40	5	21
45	5	24
50	15	14
55	8	2
60	9	2
65	17	14
70	7	7
75	5	9
80	4	18
85	2	11
90	9	3
95	19	15
100	16	6

Student 40		
Age	Gene 1	Gene 2
5	23	5
10	8	2
15	17	8
20	11	12
25	15	11
30	20	1
35	9	5
40	15	8
45	6	19
50	6	1
55	16	22
60	21	12
65	22	21
70	10	23
75	16	11
80	23	12
85	24	15
90	10	24
95	6	9
100	16	15

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Collecting the Data

Age	Number of People Who Developed Cancer		Total Number of People Who Developed Cancer	
	One Hit	Two Hit	One Hit	Two Hit
5 years				
10 years				
15 years				
20 years				
25 years				
30 years				
35 years				
40 years				
45 years				
50 years				
55 years				
60 years				
65 years				
70 years				
75 years				
80 years				
85 years				
90 years				
95 years				
100 years				

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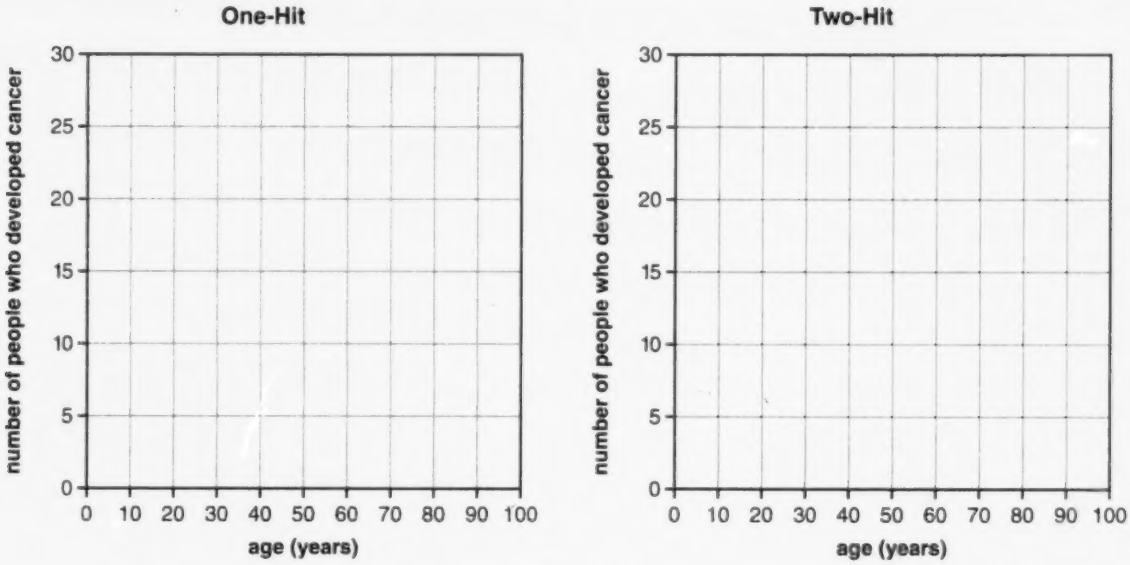
Graphing the Data

Graph the class's results for the one-hit and two-hit hypotheses, then answer the question.



Do the one-hit and two-hit hypotheses provide good explanations for the incidence of colon cancer? Explain your answer.

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Using the Hit Simulator

Follow the instructions below to use the Hit Simulator to test hypotheses about the development of cancer.

Conduct Your First Run

To familiarize yourself with the Hit Simulator, conduct a trial run as follows:

- Enter a "1" in the window labeled "number of hits required for cancer." This value means that you are testing the hypothesis that only one mutation is required for cancer to develop.
- Enter a "0.5" in the window labeled "mutation rate per age interval." This value means that you are testing the hypothesis that there is a 50 percent probability of experiencing a cancer-causing mutation at each age interval.
- Click the button labeled "Calculate Next 5 Years." The bar on the graph on the right side of the screen indicates the percentage of people in a population who would be expected to develop cancer by the age of 5 if the mutation rate were 50 percent and only one hit was required for a cell to become cancerous.
- Click the button labeled "Calculate to Age 100." The bars that appear on the graph indicate the percentage of people at each age interval who would be expected to develop cancer if the mutation rate were 50 percent and it required only one hit in order for a cell to become cancerous.

Investigate the Effect of Changing the Number of Hits Required

Use the Hit Simulator to investigate how the incidence of cancer in a population would be expected to change if different numbers of hits were required for a cell to become cancerous. For this investigation, keep the mutation rate set at 0.5 (50 percent). Conduct the runs indicated, then conduct three of your choice. Record your results in the following table.

Effect of Changing the Number of Hits Required

Number of Hits	Mutation Rate	Percentage of People Expected to Develop Cancer		
		By Age 25	By Age 60	By Age 80
1	0.5			
2	0.5			
5	0.5			
	0.5			
	0.5			
	0.5			

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1. How does the incidence of cancer change as you require a greater number of hits for a cell to become cancerous?

2. Recall the graph of the incidence of colon cancer that you observed at the beginning of this activity. Did the incidence of cancer in any of the runs you just completed match the incidence of cancer recorded in that graph? Explain your answer.

3. What can you conclude from this observation?

Investigate the Effect of Changing the Mutation Rate

Use the Hit Simulator to investigate how the incidence of cancer in a population would be expected to change if the mutation rate were different. For this investigation, keep the number of hits required set at 1. Conduct the runs indicated, then conduct three of your choice. Record your results in the following table.

Effect of Changing the Mutation Rate

Number of Hits	Mutation Rate	Percentage of People Expected to Develop Cancer		
		By Age 25	By Age 60	By Age 80
1	0.1			
1	0.5			
1	1.0			
1				
1				
1				

4. How does the incidence of cancer change as the mutation rate increases?

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5. Recall the graph of the incidence of colon cancer that you observed at the beginning of this activity. Did the incidence of cancer in any of the runs you just completed match the incidence of cancer recorded in that graph? Explain your answer.

6. What can you conclude from this observation?

Investigate the Effect of Changing
Both the Number of Hits Required and the Mutation Rate

Now use the Hit Simulator to investigate how the incidence of cancer in a population would be expected to change with different combinations of the number of hits required and mutation rates. Conduct the runs indicated, then conduct three of your choice. Record your results in the following table.

Effect of Changing Both the Number of Hits Required and the Mutation Rate

Number of Hits	Mutation Rate	Percentage of People Expected to Develop Cancer		
		By Age 25	By Age 60	By Age 80
1	0.1			
5	0.1			
7	0.1			
1	0.04			
5	0.04			
7	0.04			

7. What can you conclude from your observations?

Summary

8. What clue did the change in risk of colon cancer provide scientists about the cause of cancer?

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Testing an Explanation by Looking at Additional Data

It sometimes happens that as scientists begin to explain one thing (for example, cancer as a multistep process), they find that they also can explain other observations. In fact, an idea's power to help us explain other things we've observed gives us new evidence that the idea may be correct.

Use your new understanding of cancer as a multistep process to explain the following observations.

1. ***Cancer is a disease of aging.*** "With a handful of exceptions, cancer is a disease of aging and is vastly more likely to strike in the middle or later years than in childhood, youth, or young adulthood. Indeed, experts unanimously cite age as the single most important risk factor [for cancer]."[1]

   Explanation:

2. ***You've come a long way, baby.*** "[There was a] 20–25-year lag between the onset of widespread cigarette smoking among women after World War II [1945] and the massive increase in female lung cancer detected in the 1970s."[2]

   Explanation:

3. ***Genes and increased susceptibility.*** "If a woman carries this mutation [*BRCA1*], she faces . . . an [increased] risk—not a certainty—of developing breast cancer. . . . If a woman does not carry this mutation, her risk of breast cancer is . . . [lower]."[3]

   Explanation:

1 Murphy, G.P., Morris, L.B., & Lange, D. 1997. *Informed decisions.* Viking: The American Cancer Society.
2 Varmus, H., & Weinberg, R.A. 1993. *Genes and the biology of cancer.* New York: Scientific American Library.
3 Sidransky, D. 1996. Advances in cancer detection. *Scientific American, 275*(3): 104–109.

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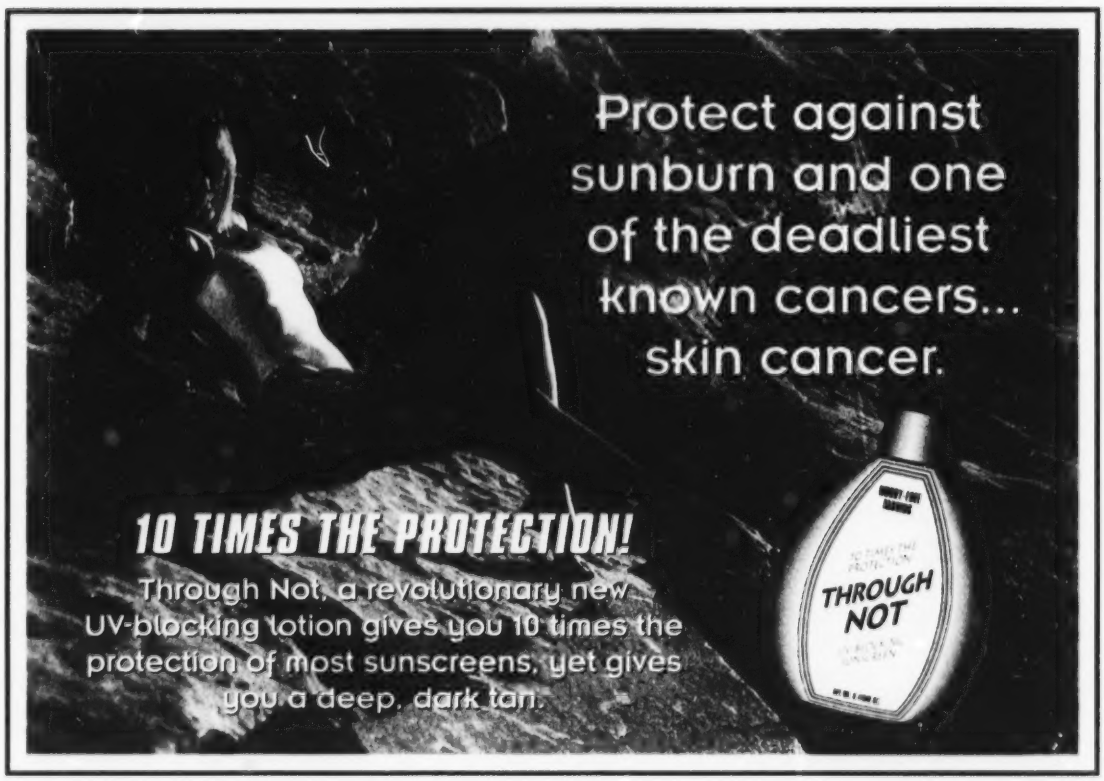
Media Item 1

Work with your teammates to review this media item. First, identify the claims the item makes about the product, ultraviolet light, and cancer. Then, describe the evidence on which these claims are based.


Protect against sunburn and one of the deadliest known cancers... skin cancer.
10 TIMES THE PROTECTION!
Through Not, a revolutionary new UV-blocking lotion gives you 10 times the protection of most sunscreens, yet gives you a deep, dark tan.
THROUGH NOT

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Media Item 2

Work with your teammates to review this media item. First, identify the claims the item makes about the product, ultraviolet light, and cancer. Then, describe the evidence on which these claims are based.

Are you risking eye damage every time you go out in the sunlight?

RECENT SCIENTIFIC DISCOVERIES PROVE THE EXISTENCE OF HARMFUL ULTRAVIOLET RAYS, EVEN IN NATURAL SUNLIGHT. PROTECT YOUR EYES FROM THE HARMFUL EFFECTS OF THESE RAYS WITH NEW VISION UV-BLOCKING EYEWEAR – THE FINEST IN PROTECTIVE EYEWEAR.
NEW VISION PROTECTIVE EYEWEAR

New Vision UV-blocking eyewear – available at finer retailers nationwide. Learn more on our web site: www.newvision.com.

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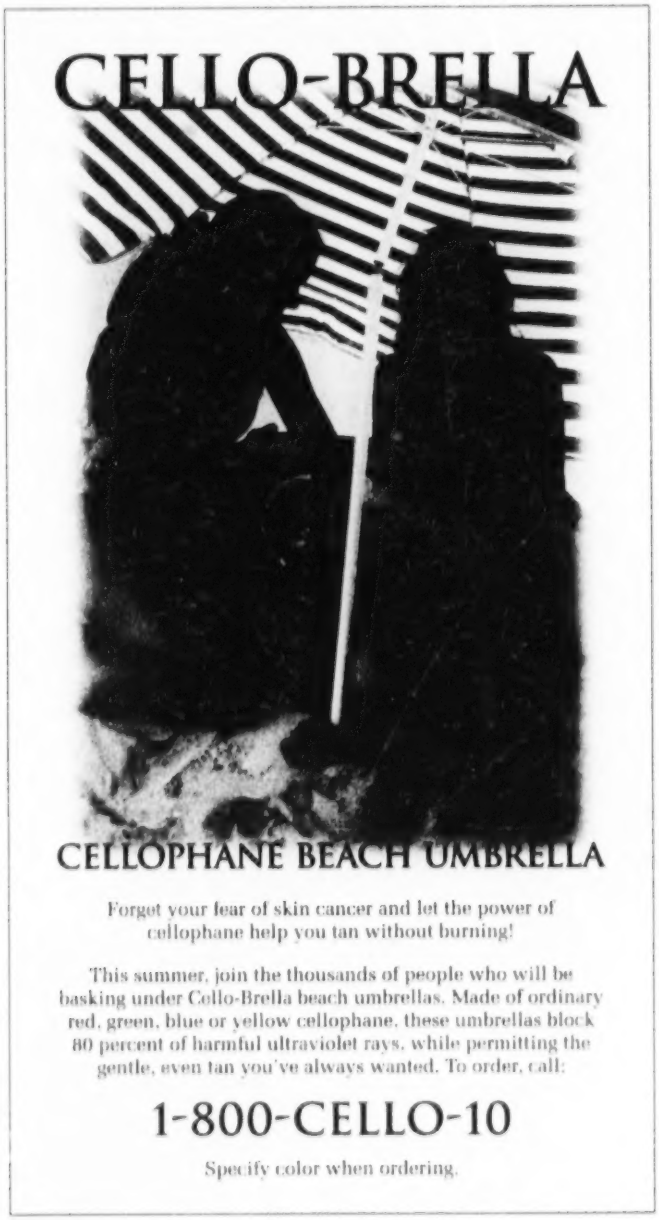
Media Item 3

Work with your teammates to review this media item. First, identify the claims the item makes about the product, ultraviolet light, and cancer. Then, describe the evidence on which these claims are based.


CELLO-BRELLA
CELLOPHANE BEACH UMBRELLA
Forget your fear of skin cancer and let the power of cellophane help you tan without burning!
This summer, join the thousands of people who will be basking under Cello-Brella beach umbrellas. Made of ordinary red, green, blue or yellow cellophane, these umbrellas block 80 percent of harmful ultraviolet rays, while permitting the gentle, even tan you've always wanted. To order, call:
1-800-CELLO-10
Specify color when ordering.

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Media Item 4

Work with your teammates to review this media item. First, identify the claims the item makes about the product, ultraviolet light, and cancer. Then, describe the evidence on which these claims are based.

No more tan lines!
Get an all-over tan...right through your swimsuit.
Our revolutionary new MicroPore fabric takes the worry out of sunbathing:
No unsightly tan lines.
Protects against sunburn.
Prevents skin cancer.
Available in over 100 beautiful styles for men and women.
Call 1-800-555-4TAN or see us on the web at
SOLARIS
SWIMWEAR

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Using a Model System to Test Claims About UV Light

You and your teammates will use yeast as a model system for testing a claim your media item made about a particular product and UV light. Follow the steps below to design and conduct a controlled experiment that tests such a claim.

Learn About UV Light and Yeast

1. Read the following paragraphs to learn about ultraviolet (UV) light and the yeast you will use to test claims about UV light.

   What is UV light?
   UV light is one of the forms of radiation that is produced by the sun and by a variety of other sources (for example, certain types of artificial lights). UV light is not visible to us, but it is all around us. It is the part of sunlight that causes sunburns and tans. It also is the part of sunlight that can cause skin cancer.

   Why is UV light dangerous?
   UV light can damage the DNA inside cells. Cells repair most of this damage, but occasionally a cell makes a mistake during this repair process. This mistake causes a mutation in one of the cell's genes. The accumulation of mutations inside skin cells can lead to skin cancer.

   What are yeast?
   Yeast—in this case, baker's yeast (scientific name, *Saccharomyces cerevisiae*)—are a simple, single-celled form of fungus. Yeast reproduce both sexually and asexually and have simple nutritional needs. You need a microscope to see a single yeast cell. But that cell can grow into a whole colony of cells (that you can just barely see) in one day if it is provided with the right conditions.

   Why are yeast a good organism for testing claims about UV light?
   Yeast are easy to grow in the laboratory. The type of yeast that you will use is especially sensitive to UV light. It cannot repair the damage that UV light causes to its DNA. Thus, these yeast are killed by sunlight. As you will see, you can use these UV-sensitive yeast to measure the killing effect of sunlight under different conditions.

   Human skin cells (as well as normal yeast cells and most other normal cells) have enzymes that repair damage to DNA that is caused by UV light. But when too much damage occurs (as might occur, for example, when a person spends a great deal of time outdoors), the repair enzymes may not be able to keep up. Across time, mutations may accumulate inside skin cells, leading to cancer. UV-sensitive yeast are a good **model** for testing products claimed to protect a person from skin cancer because the yeast's sensitivity allows the damaging effects of UV light to be observed very quickly.

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Write a Hypothesis

2. Write the claim you would like to test about UV light.

> Be sure that your claim is related in some way to your media item. An example of a claim about UV light is that the higher the altitude on the earth's surface, the greater the amount of UV light present.

3. Now write the claim that you want to test in the form of a question.

> If you wanted to test the claim that more UV light reaches the earth's surface at higher altitudes than at lower altitudes, you might ask, "Does more UV light reach the earth's surface at higher altitudes than at lower altitudes?"

4. Write a tentative answer to your question.

> Scientists call such tentative answers "hypotheses." Your hypothesis about UV light and altitude might read, "Yes, more UV light reaches the earth's surface at higher altitudes than at lower altitudes."

5. Use your hypothesis to write a prediction.

> A prediction is a sentence that describes something that would happen if your hypothesis is correct. For example, your prediction about UV light and altitude might read, "If the amount of UV light that reaches the earth's surface increases with increasing altitude, then the maximum at altitude X will be greater than the maximum at altitude Y."

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Design an Experiment

6. Describe the major parts of your experiment.

- What are your variables? That is, what will you change? What else might change?

> In the UV light and altitude experiment, you would need to change the altitude at which you measure the UV light coming from the sun. The amount of UV light might also change as a result of the difference in altitude.

- What will you measure?

> In the UV light and altitude experiment, you would measure the altitude and the maximum amount of UV light received in a certain size area during a certain length of time.

- How will you measure this?

> In the UV light and altitude experiment, you might use published values for altitude, and you might measure the ability of UV light to kill UV-sensitive yeast as an indication of the amount of UV light received during a certain period of time.

- What is your control?

> A control is a group of individuals in an experiment that do not receive the treatment given to the test subjects. In the UV light and altitude experiment, you might prepare a set of identical plates and expose half to UV light at the two altitudes, but leave the other half unexposed. The unexposed plates are your controls.

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7. Write a description of your experiment and submit it to your teacher for approval. Make any changes your teacher suggests.

Conduct the Experiment

8. Follow the instructions below for spreading your plates of yeast and conducting your experiment.

   Collect the following materials:

   - culture of G948-IC/U yeast (1 per team)
   - sealed tube containing 10 to 15 ml of sterile water (1 per team)
   - packet of sterile toothpicks (1 per team)
   - 1-ml sterile calibrated bulbed transfer pipet (1 per team member)
   - petri plate containing YED agar medium (1 per team member)

   Your teacher will indicate other materials that you may need to test your claim.

   a. Open one end of your packet of toothpicks and carefully remove just one toothpick. (**Do not touch the other end of the toothpick.**)

   b. Make a visibly turbid yeast suspension by using the toothpick to scrape some yeast from your culture plate and wiping them off on the inside of your tube of sterile water. Replace the lid and swirl to mix. If the suspension is not visibly cloudy, add more yeast cells, using a new toothpick.

   c. Swirl the tube to resuspend the cells before removing each sample. Remove the sterile pipet from its wrapping just before you use it and carefully remove 1 ml of the yeast suspension. (**Do not touch the end of the pipet.**)

   d. Lift the lid of a YED agar plate at an angle just enough to deposit the 1 ml of the suspension directly onto the surface of the plate. Replace the lid of the plate.

   e. Tilt and rotate the plate to spread the cells over the surface of the agar. If the liquid does not cover, use the blunt end of a toothpick to spread the suspension.

   f. Let the agar absorb the liquid until it disappears (about 10 minutes).

   g. Secure the lid to the bottom of the petri dish using a small piece of tape on the side of the dish. (The plastic lid of the petri dish does not absorb a significant amount of the UV light found in sunlight, but the tape will.)

   h. Treat your plates according to the experimental plan you devised. Then, if appropriate, expose the plates to direct sunlight outdoors for the number of minutes given in the table below.

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Exposure Times for Yeast Plates

Time of Year	Midmorning (Minutes)	Noon (Minutes)	Midafternoon (Minutes)
summer	3–4	2–3	3–4
spring and fall	5–6	3–4	4–5
winter	40–50	15–20	20–30

i. Incubate your plates **in the dark**, overnight at 30°C, or two days at room temperature. Incubate with the agar side up to prevent condensation from dropping on the colonies.

Record the Results

9. Collect your plates from the previous day and describe in writing and/or sketch the results on each plate.

Report the Results

10. Present your results in the following manner:

a. State the claim you tested.

b. Explain how you tested the claim. (For example, What did you change? What did you measure?)

c. Describe your results.

d. State your conclusion(s).

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Evaluating Claims About Cancer

Follow the steps below to evaluate another team's media item.

1. **Identify the claims.**

   Ask yourself, "What are the explicit (obvious, stated) and implicit (less obvious, not stated directly) claims being made here?"

2. **List the evidence.**

   Ask yourself, "What evidence is offered to support each of these claims?"

3. **Evaluate the evidence.**

   Ask yourself questions such as the following:

   - What is the source of the evidence?

   - Who did the experiment? Who funded it? Is there any reason to think the results might be biased?

   - How many subjects were in the study? Were there proper controls?

   - Are there other reasons the researchers might have obtained these results?

   - What other ways could these claims be tested? Is there any way I can test these claims?

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- Where else could I find reliable information about this topic?

4. **Evaluate the claim.**

   Ask yourself whether, based on the evidence, you accept the claim, accept it tentatively until you can learn more about it, or reject the claim.

5. **If appropriate, act on the basis of your evaluation.**

   Ask yourself what you should do, based on the outcome of your evaluation.

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A Proposed Statute

Mandatory Use of Skin Protection for All Individuals Under the Age of 18

Whereas it is well documented that only 15 percent of Americans regularly wear a sunscreen when they are outside, and 25 percent never wear sunscreen.

Whereas there is a direct link between the sun's ultraviolet (UV) rays and melanoma, the deadliest form of skin cancer.

Whereas there were more than 42,000 new cases of malignant melanoma diagnosed in 1999.

Whereas more than 7,000 Americans die each year from melanoma.

Whereas disruption of the earth's ozone layer by atmospheric chemical pollution may lead to rising levels of UV radiation.

Whereas 80 percent of a person's UV exposure occurs prior to age 18.

Be it enacted by the Federal Statutes that:

All individuals under the age of 18 are required to wear headgear and clothing that covers 90 percent of the extremities while outside during peak hours of UV exposure. This covering shall occur in all public locations that are currently under federal jurisdiction, including public school property, recreation sites, federal buildings, and work sites supervised by employers that are overseen by OSHA regulations.

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Getting Prepared to Support or Oppose the Statute

Follow the steps below to develop your list of reasons to support or oppose the proposed statute.

1. Spend about 5 minutes in a brainstorming session identifying reasons to support the statute and reasons to oppose it. Fill these reasons into the table on page 5.2b.

2. View the video clips on the CD-ROM (*A Proposed Statute* and *The People Respond*) that show people commenting on the proposed statute. What questions do these people raise? Add these issues to the table.

3. Ask yourself what additional information about UV light and skin cancer might help strengthen your position. For example, you may wish to look for evidence to support the reasons you have listed or for information that can help you answer the following questions:

   - What is skin cancer? Who is most at risk? What outcomes can people who develop skin cancer expect? What outcomes does society experience as a result of skin cancer?
   - How can people reduce or prevent dangerous exposure to UV radiation? How effective are these different methods of protection?
   - Is UV exposure really a risk factor related to skin cancer? When and where does most exposure occur? Are there other important sources of UV exposure?
   - Are there other cases where society has limited behavior for public health reasons? For example, what can we learn from the Australian experience with skin cancer? Are there other examples of limiting behavior for public health reasons? How effective are they?

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Reasons to Support or Oppose the Statute

To Support the Statute	To Oppose the Statute

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Analyzing the Results of a Public Policy Discussion

Answer the following questions related to the public policy discussion you just completed.

1. What revisions, if any, would you make to the statute in the light of the reasons you heard?

2. What other suggestions can you make about reducing the incidence and impact of skin cancer in the United States?

3. How does this activity illustrate that
   - good choices can reduce a person's chance of developing cancer?
   - values sometimes conflict in debates about laws related to personal and public health?
   - it is possible for people to hold different positions on a controversial topic and still participate in a reasoned discussion about it?

4. How has research about cancer helped improve personal and public health in the United States? Answer specifically, using examples drawn from all five of the activities in this module.

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